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SURGERY

A Monthly Journal Devoted to the Art and Science of Surgery

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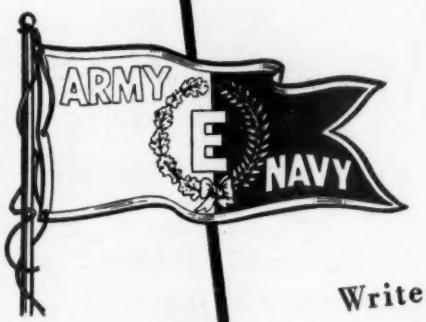

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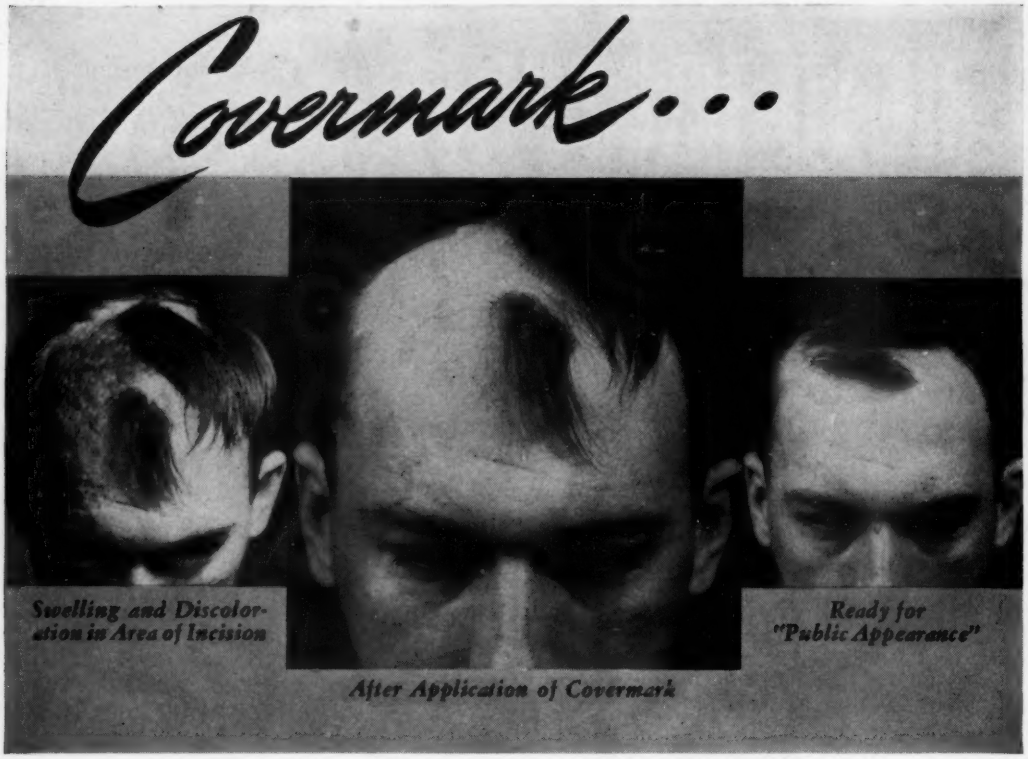
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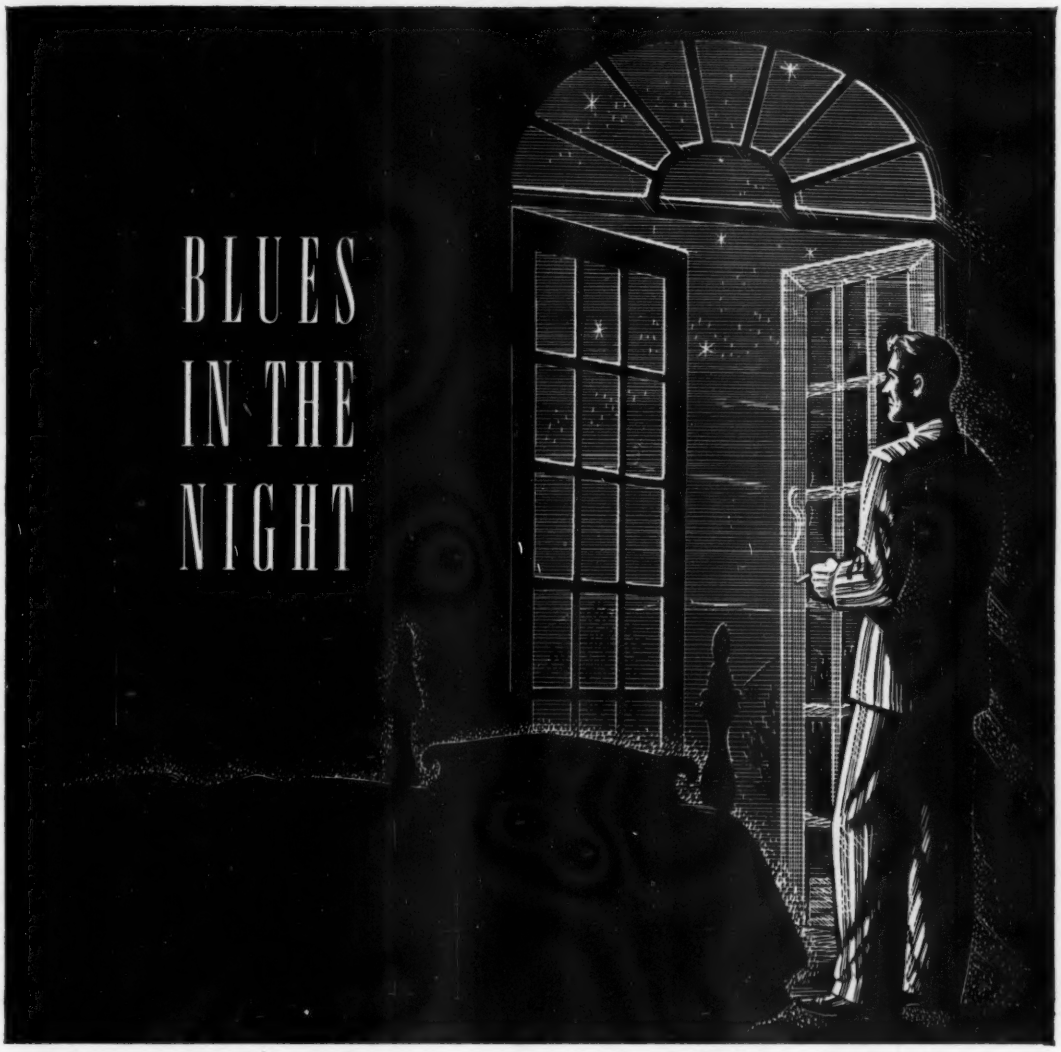

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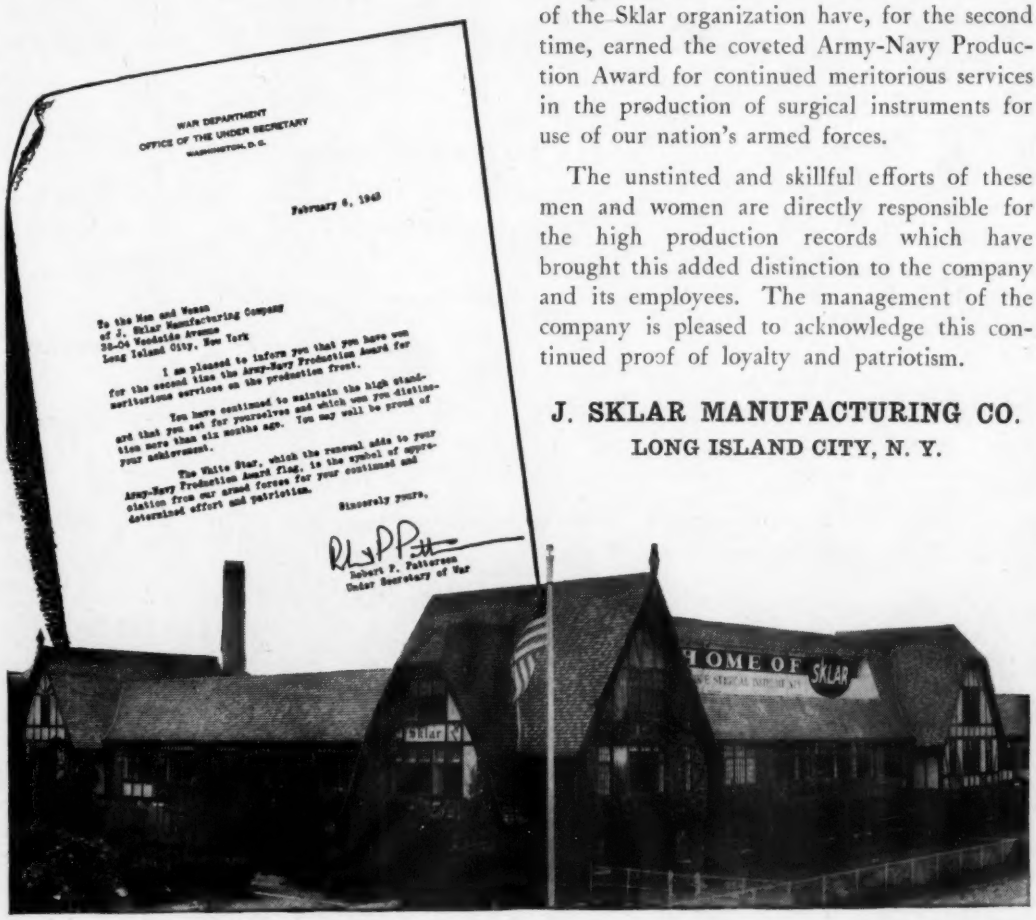

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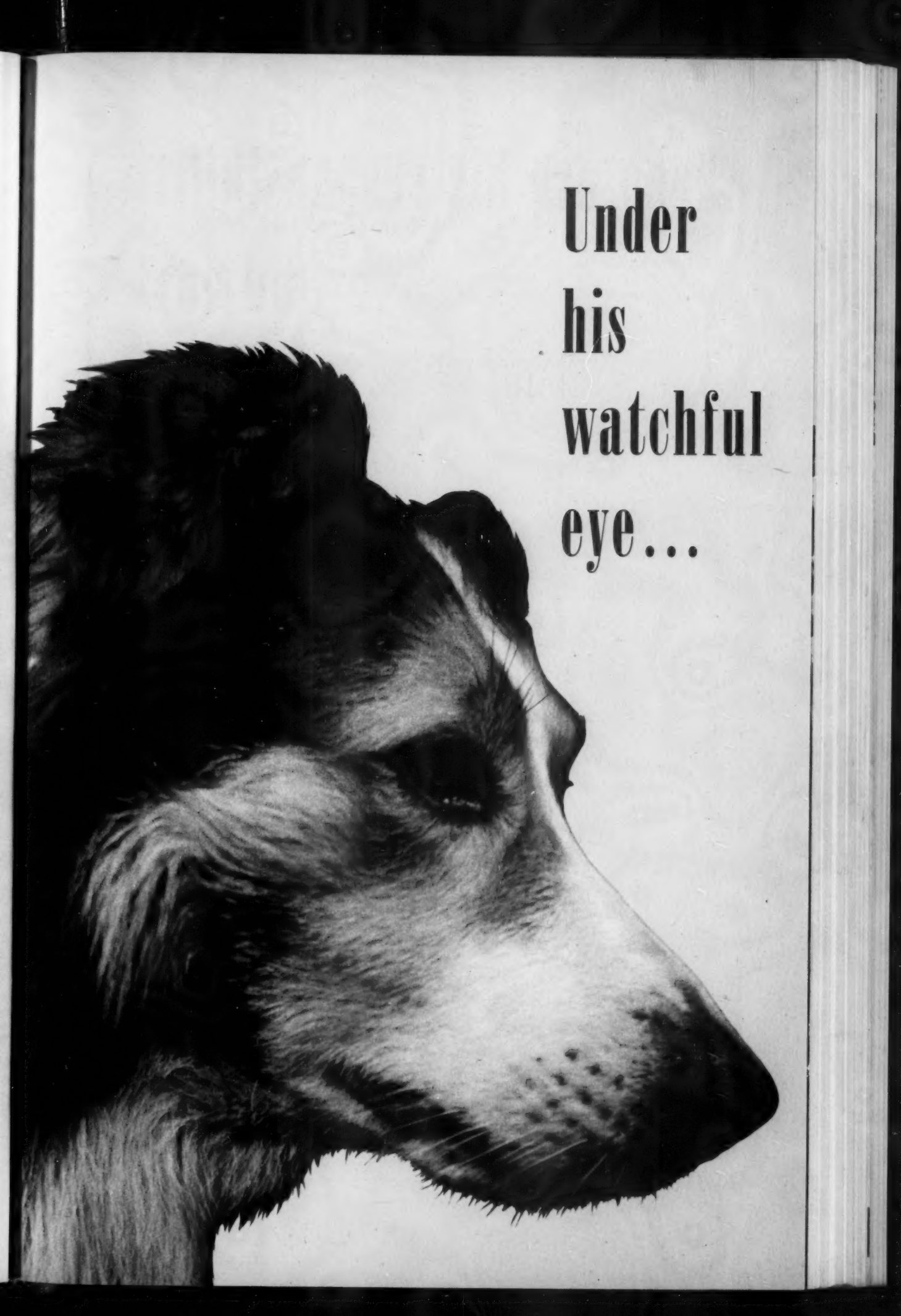
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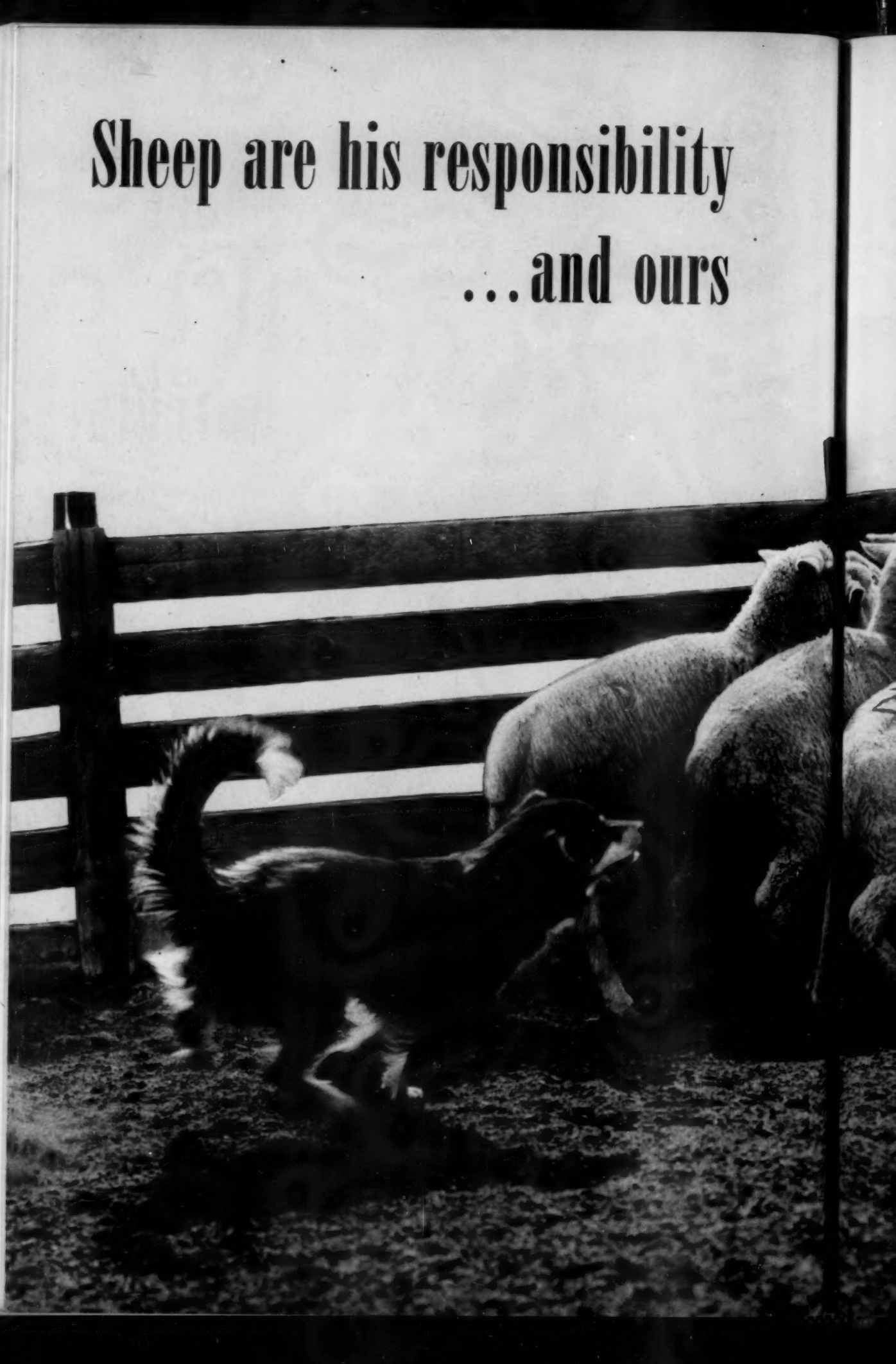

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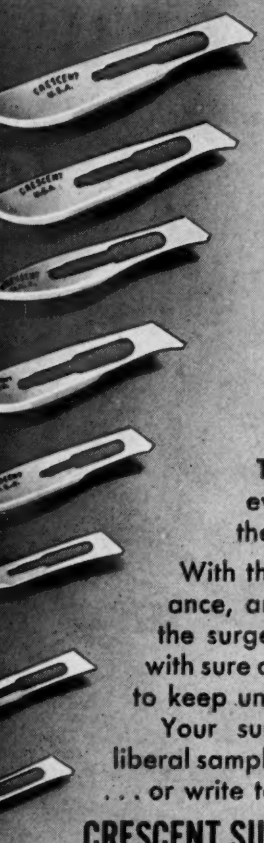

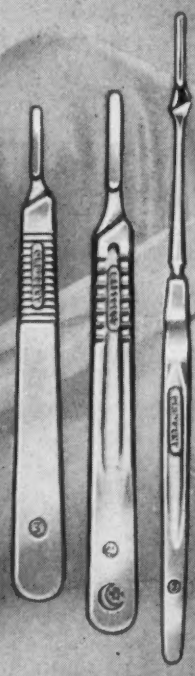

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SURGERY

VOL. 13 APRIL, 1943 No. 4

Original Communications

THE PHYSIOLOGIC BASIS FOR THE SURGICAL TREATMENT OF ASTHMA

LAURENCE MISCALL, M.D., AND E. A. ROVENSTINE, M.D.
NEW YORK, N. Y.
(From the Second Surgical Division and the Division of Anesthesia, Bellevue Hospital)

FOR the purpose of this discussion asthma is defined as an abnormal type of respiration with labored breathing due to obstruction of the conducting airway system. Respiration is the total process of gas interchange, with breathing only one of its several related physiologic mechanisms. Respiration continually changes to meet the varying needs of normal life or to accommodate for the malfunction introduced by disease. Active inspiration and passive expiration suffice for normal quiet respiration and breathing is adjusted to greater normal demands by the chemical and reflex control of muscular activity. If the demand exceeds markedly the ability to ventilate, or the respiratory efficiency is embarrassed by either intrinsic or extrinsic pulmonary factors, labored breathing occurs. In asthma the factor is intrinsic widespread obstruction of the conducting airway system. Moreover, in some types of asthma this alteration in the normal function of the bronchial muscle and mucosa is initiated by excessive activity of reflexes which follow definite nerve pathways. When these reflexes originate in the lung, the afferent pathway traverses the sympathetic nervous system and may be interrupted surgically as the therapeutic measure.

THE RELATION OF STRUCTURE AND FUNCTION

The transition from terminal to respiratory bronchiole separates the proximal conducting from the distal exchange division of the lung. Their anatomic characteristics are distinctly related to function in abnormal as well as normal respiration. The epithelium gradually shifts from pseudostratified columnar in the trachea, and ciliated columnar in the terminal bronchiole, to nonnucleated respiratory plates in and

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beyond the alveolar ducts. Mucous glands, numerous in the trachea, are absent in the bronchioles. The bronchial muscle embraces the mucosa from the trachea to the alveolar mouths and despite an actual decrease, relatively increases by the fading out of other constituents. It is most prominent in the terminal bronchiole where the lumen is narrowest and unsupported by cartilage. Distally, as elastic tissue steadily replaces muscle, it changes to a myo-elastic layer. The tunica propria contains blood vessels, nerves, elastic fibers, wandering cells, and lymphocytes. The bronchial artery supplies the conducting, and the pulmonary artery the exchange division. The bronchial artery exhibits the typical innervation and reactions of the greater circulation of which it is a part.

The respiratory center integrates all respiratory functions. This formation of fibers and motor cells in the pons and upper medulla is especially fitted to correlate impulses from many areas. It has been divided into the pontile pneumotaxic, the inspiratory, and the expiratory (inhibito-inspiratory) centers. Impulses from the respiratory center adjust the vigor and rate of respiration by virtue of three characteristics; an automatic rhythm subject to modification by nervous and chemical factors; graded sensitivity to chemical stimuli; reciprocal innervation of inspiratory and expiratory centers. Automatic rhythm is established by chemical and nervous impulses inducing instability in one or several cells in the inspiratory nucleus. So-called "toppling" results in a burst of efferent impulses causing sustained inspiratory muscular contraction which Lumsden called apneusis.[1] Apneusis does not occur unless impulses which inhibit inspiration normally are excluded by vagotomy and pontile section.[2] These impulses follow two pathways. Those from the pulmonary stretch receptors, stimulated by expansion, pass in the vagi to the expiratory center and from there spread out to inhibit the inspiratory center. The original efferent discharge sends impulses to the pontile center which spread out to inhibit the inspiratory center and possibly excite active expiration. Thus, the center has no inherent rhythm but one dependent upon inhibition by either lung reflexes set up by expansion or reflexes from the pontile pneumotaxic center excited by the original inspiratory center discharge. Graded sensitivity of the respiratory center results either from the activity of a variable number of cells or from changes in synaptic resistance. This capacity may institute any activity from quiet respiration to dyspnea by quantitative control of muscular stimulation. Reciprocal innervation affects both phases of respiration. As impulses appear in inspiratory fibers they disappear in the expiratory and vice versa.[3] Pitts, Magoun, and Ranson[4] considered the expiratory center fundamentally inhibito-inspiratory after they noted that electrical stimulation in the medulla (of cats) usually inhibited inspiration but active expiration followed excessive stimulation only. Expiration becomes active when chemical or nervous stimuli are sufficiently intense and after the expiratory center is released from the inhibition of inspiration.

The respiratory center is affected by numerous chemical and nervous stimuli. It is so specifically sensitive to carbon dioxide that hyperpnea follows any slight increase in its concentration. Anoxemia, however, causes hyperpnea primarily through chemoreceptor reflexes rather than by central action. Although these reflexes do not regulate normal respiration since anoxemia is rarely severe enough, they may reflexly drive it in abnormal states when circumstances are sufficiently adverse to directly depress the center. Such hyperpnea may further impair the functional capacity of its neurons already depressed by local hypoxia. The consequent reduction of blood carbon dioxide lowers specific stimulation and contributes to cerebral vasoconstriction. Although carbon dioxide normally regulates respiration, intra- or extrapulmonary reflexes are important since they may alter the respiratory pattern without evident blood chemical changes. While the Hering-Breuer reflexes and those reflexes from aortic and carotid zones are considered essential to the automatic control of respiration, others attain importance only in defective states like asthma. Definite anatomic pathways and physiologic effects have been demonstrated frequently for these respiratory reflexes. The sympathetic and parasympathetic divisions of the automonic nervous system each contribute both afferent and efferent elements to the lungs where Ranson and Billingsley[5] found a wide variety of nerve structure.

Parasympathetic afferents run centrally in the vagus to the nodose ganglia, which most writers agree contain the cell of origin. Chase and Ranson[6] noted that these typically large sensory fibers disappeared from the vagus below the pulmonary rami. By vagotomy and degeneration, Larsell and Mason[7] showed that these came principally from the homolateral lung. Following the vagus rootlets and fasciculus solitarius they terminate in the nucleus of the tractus solitarius on cells which send fibers to the motor nuclei over the reticular formation. A reflex involving this pathway would traverse three neurons: a primary visceral afferent of the vagus; a secondary afferent with its cell in the nucleus of the tractus solitarius; a visceral efferent with a cell in the visceral efferent column.[6] Head's[8] study of the distribution of sensory spinal fibers through sympathetic pathways placed the lung supply from the upper five thoracic nerves. These originate in cells in the dorsal cerebrospinal ganglia,[9, 10, 11] not the sympathetic, and enter the sympathetic chain mainly in the upper three white rami.[12] Möllgaard[13] outlined their course over the vagus, ansa subclavia, stellate ganglia, and white rami to cells in the cerebrospinal ganglia of the upper three thoracic nerves. Their central processes pass to the dorsal horn and ascend or descend in the cord. Additional definite evidence indicates that many sympathetic afferent fibers reach the chain either directly or over the cardiac accelerator nerves.[14, 15] This pathway is easily traced and does not involve the cervical sympathetic trunk.[11] The lack of degeneration after vagotomy supports the sympathetic origin of these fibers which

traverse mainly the stellate, second and third thoracic ganglia, and white rami in reaching the cord.[7] Efferent parasympathetic fibers originate in the dorsal vagal nuclei and follow the vagus through the nodose ganglion to the lungs as preganglionic fibers. Larsell and Mason,[7] after vagotomy in rabbits, concluded that the majority of this motor supply was homolateral with minor cross-over in the posterior pulmonary plexus. Möllgaard[13] considered all of these efferent fibers either bronchomotor or secretory in function and further subdivided the bronchomotors into constrictors or dilators. This division is definitely open to question. The efferent sympathetic fibers from the intermediolateral cell column pass as preganglionic fibers via the white rami of the upper five thoracic nerves to the sympathetic chain. Möllgaard[13] believed that these fibers reached the vagus of the same and opposite side through the stellate ganglia and ansa subclavia and were vasomotor only. However, it has been proved that these fibers reach the lung directly or over the accelerator nerves and that they are not entirely vasomotor in function.[15] Rasmussen has supplied a diagram which graphically illustrates a conception of some of these pathways.[16] Larsell[17] has carefully described the sensory pulmonary terminations of these nerves and closely linked their location and structure to function. For example, at the division of the bronchi, bulbous nodules seem responsive to tactile irritation. In the respiratory bronchioles and alveolar ducts the thickened epithelial nodules seem fitted to react to the pressure stimulus of collapse. The most distal thinly encapsulated receptors in the alveolar walls and mouths are sensitive to distention. The sensory endings initiate all reflexes of pulmonary origin traveling the pathways already enumerated. The typical arrangement of preganglionic motor fibers to ganglia and postganglionic fibers terminating on smooth muscle cells, described by Ploschko[18] for the trachea, applies to the bronchi also. Clusters of ganglion cells along the bronchi contain large nerve trunks having myelinated and unmyelinated fibers. Although most motor fibers are postganglionic, some leave the nerve trunks and go directly to muscle cells near the nuclei. It is not unlikely that these originate in cells at some distant source outside the lung. Since ganglion cells have not been found beyond the larger bronchi, the nerve bundles must carry sensory and both motor and inhibitory fibers. This anatomic variation closely parallels the marked difference in physiologic type of fibers found by Carlson and Luckhardt[19] in the pulmonary nerves of different animals.

Extensive experimental evidence indicates that impulses over these and other pathways markedly affect respiration. Those normal impulses which involve the pulmonary conducting system primarily are almost exclusively autonomic in course and reflex in type. Their hyperactivity is the basis of asthma. By section and stimulation of nerve endings, Dixon and Brodie[14] and Dixon and Ranson[15] outlined these reflexes which exert both a primary peripheral and a secondary central respira-

tory effect. They found with other factors constant that ventilation was measurably altered by any change in either the elasticity of the lungs or the resistance offered by the trachea, bronchi, or alveolar ducts. To stabilize elasticity, they eliminated variations in capillary blood, effusion, and secretion and studied the element of resistance. Muscular contraction appeared much more effective than mucosal edema and congestion in constricting the conducting lumen and increasing the resistance. By recording the changes in intratracheal pressure, airwave amplitude, and the phasic rate of respiration, they collected fundamental information on these reflexes which affect the lungs directly. Bronchoconstriction which decreases breathing, followed excitation of the distal ends of the cut vagus by stimulating motor constrictor fibers. These fibers, which originate in the dorsal vagal nucleus and supply the lungs over both vagi, are mainly homolateral in distribution but may cross completely. Similar constriction followed excitation of these endings by the parasympathomimetic drugs, muscarine and pilocarpine. Constriction is absent after nerve degeneration or atropinization. Vagal stimulation relieved constriction from pilocarpine by exciting a dilator reflex afferent in the vagus and efferent in the sympathetics.[15] Their conclusion that this vagal constricting action exerted no tonic effect on the bronchi may not be justified. These impulses induce a return of tonus during normal expiration and are significantly hyperactive in asthma.

Dixon and Ranson[15] studied the bronchodilator nerves in their reinvestigation of the subject. Bronchial dilatation which increases breathing almost invariably followed stimulation of the peripheral ends of cut sympathetic fibers distal to the stellate ganglion. This contrasts sharply with the constriction from similar vagal stimulation. The motor dilating fibers, very few of which travel in the vagus, originate in the intermediolateral cell column and traverse predominantly the upper white rami to the sympathetics and stellate ganglia. Similar dilatation follows adrenalin excitation of the motor dilator endings or atropine paralysis of the motor constructor endings.

These efferent components have been linked to definite afferent arcs to complete the patterns of bronchial reflexes. Reflex bronchodilation followed stimulation of afferent fibers in the central ends of the sectioned vagi. This afferent impulse traverses the vagus to the medulla, descends in the lateral columns, and leaves the cord as an efferent impulse over the white rami of the upper three thoracic nerves. It reaches the lungs via the stellate ganglion but not entirely through the cervical sympathetic or cardiac nerves, since their section does not abolish it but high cervical cord section does. Stimulation of the central end of the cut accelerator nerves produces reflex constriction by impulses afferent in them and through the cervical sympathetics, stellate ganglia, and cord to the medulla. They excite, in motor nerve cells here, constrictor impulses efferent in the vagus. The vagus also contains a few afferent elements, the central stimulation of which causes bronchoconstriction.

These impulses are afferent and efferent in the vagus, section of which on the opposite side abolishes the reflex by interrupting the efferent limb. The profound significance of this mechanism is emphasized in the later discussion of sympathetic nerve block.

Numerous impulses which do not influence the respiratory cycle by any action on the pulmonary conducting system arise in widely separated areas. They are not exclusively autonomic in course but may follow such pathways. Their hyperactivity is a contributory factor in asthma. Head,[20] in 1889, recorded the contractions of the diaphragm as a means for investigating such afferent vagal impulses. Vagotomy induced a long contraction (inspiration) from increased tonus of the diaphragm. The chest assumed the inspiratory position with slower and deeper breathing which quickly returned to normal with the characteristic response to carbon dioxide. The increased tonus wore off or was lost after section of the efferent pathway in the phrenic nerves. Vagotomy temporarily increased the strength and duration of tonic and rhythmic inspiratory effort by obliterating the activity of normal vagal impulses which actually depress inspiratory effort. Similar reflex effects are conducted over extravagal paths. Gault and Scott[21] found that double vagotomy slowed respiration and prolonged inspiration, while low cervical cord section merely prolonged expiration. Combined, they lengthened both phases with slowing of respiration. Coombs[22] noted a decrease or cessation of costal respiration with a change in rhythm after dorsal root section. Coombs and Pike[23] observed that the slower respiration of double vagotomy was equally altered by section of either the dorsal roots or the corpora quadrigemina at the inferior colliculus. They, like others, concluded that the intercostal muscles excited afferent impulses which ascended over the dorsal roots and cord to the mesencephalon. However, it must be realized that dorsal root section also interrupts important afferent pathways from the lungs.

Head[20] clamped the trachea in different phases of breathing and restudied the reflexes first described by Hering and Breuer in 1868. Lung distention inhibited diaphragmatic contraction (inspiration) with a cause normally occupied by relaxation (passive expiration). This pause was broken by a longer and stronger inspiration induced by the mounting carbon dioxide of asphyxia which overcame the nervous inhibition. Pulmonary collapse provoked a normally spaced inspiratory contraction. This was strong and prolonged since the clamped trachea prevented lung distention which normally inhibits inspiration reflexly. These reflexes changed quantitatively with extremes in either phase. The impulses from overdistention inhibited inspiratory contractions much longer than normal. This was not due to overventilation since the use of pure hydrogen did not alter it. Abnormal collapse produced by sucking air from the trachea caused immediate and vigorous inspiratory contractions. Head[20] visualized the vagus as the afferent pathway since its section obliterated these reflexes. Barry,[24] using similar methods, felt

that the inspiratory stimulus from the collapsed lung did not travel in the vagus. In the cat with the vagi and stellate ganglia excised, he found a definite decrease in the urge to inspiration. Moreover, it was completely abolished by cervical cord section but unaffected by vagotomy. He concluded that the impulse to inspire followed two routes to the cord. Those from the chest wall followed the somatic nerves, but those of pulmonary origin were transmitted over the sympathetics, stellate ganglia, and through the white rami to the upper three dorsal roots mainly. Both continued in the cord to the respiratory or other centers.

Larsell and Burget[25] elicited a forced type of expiration with polypnea by stimulating the tracheal and bronchial mucosa. The expiratory muscles were fully activated by a reflex which they believed was afferent in the vagus since its section abolished the response. Similar reflex activity involving both expiratory effort and bronchial constriction is commonly seen during bronchoscopy. Craigie,[26] with similar methods, obtained spasticity and irregular apnea from inspiratory inhibition. Finding vagotomy without effect, he concluded that these afferent impulses rose over the sympathetics and cord. Similar stimulation of the mucosa of the nose, pharynx, larynx, and trachea reflexly inhibited expiration over pathways in the trigeminal, glossopharyngeal, superior, and recurrent laryngeal nerves.

Normal respiration is the activity integrated by the normal impulses over these pathways. Impulses from the center efferent in the somatic nerves stimulate the inspiratory and inhibit the expiratory muscles. The tonus of the bronchial muscles likewise is inhibited during inspiration by efferent impulses over the sympathetics. It is restored during expiration by the uninhibited constricting impulses efferent in the vagus. This nervous control of bronchial tonus, which Macklin[27] considered reflex, changes the diameter and length of the bronchi and bronchioles. Thus, the capacity of the conducting system alternately increases with inspiration and decreases with expiration, and the major portion of the tidal air and vital capacity depends upon the maintenance of this flexibility. Relaxation permits adequate filling of the conducting portion. Tonic contraction diminishes vitiation of the next inspiration because it smoothly reduces this dead space without trapping alveolar air. Since the cross-section of successive branches of the conducting system is always larger than the parent stem, even with a decrease in individual size, and since the caliber of the exchange ducts steadily increases, the air current gradually slows. This slowing in the expanded conducting system aerates the alveoli and aids simple gas diffusion. However, the flexibility of the conducting system not only controls aeration, but also regulates the number of alveoli open for aeration. Since the capacity of the respiratory division depends primarily upon the number of alveoli aerated rather than upon expansion and contraction, adequate blood gas interchange, although a function of the respiratory division, depends

directly upon the conducting division. As the lungs distend, the sensory stretch receptors activate afferent vagal impulses which normally end inspiration by inhibiting it. Expiration occurs passively in this inhibited phase. In the hyperpnea of exercise the entire lung dilates temporarily, which, in the conducting system, increases midcapacity as the loss of bronchial excursion decreases vital capacity. In the respiratory portion the residual air increases because a maximum number of alveoli is maintained open. The retention of flexibility has, therefore, a direct effect on pulmonary efficiency.

ASTHMATIC RESPIRATION

In asthma gross malfunction occurs as abnormal impulses are initiated in the normal pathways. These abnormal impulses cause the replacement of normal bronchial flexibility by abnormal bronchial obstruction which in the conducting airway system is principally an expression of altered bronchial muscle function. Excessive stimulation of sensory endings in the bronchi excites afferent impulses which travel to the cord in the sympathetics either directly or via the stellate ganglia. After ascending in the cord to the region of the fasciculus solitarius, they stimulate in vagal fibers the efferent impulses of broncho-constriction. Irritation of other sensory areas may initiate similar reflex constriction. From the mucosa of the nose, pharynx, and larynx afferent impulses follow the olfactory bulb, glossopharyngeal, superior, or recurrent laryngeal nerves, but the efferent pathway remains in the vagus. The intensity of sensory irritation and the sensitivity of the receptors influence the degree of obstruction by controlling the number of the efferent constrictor discharges. Since these do not suffice to explain the relief observed after unilateral block or operation, it appears that some threshold of central afferent stimulation must be exceeded before constriction becomes evident clinically. With sufficient intensity abnormal spasm may result, and at the terminal bronchiole where constriction is normally greatest, it may partially or completely obstruct the airway. Incomplete relaxation reduces the ease and extent of inspiration. In expiration excessive tonus traps air distal to this point in the respiratory portion of the lungs, and the residual air rises. Thus in inspiration the tonicity of the bronchi with little flexibility reduces ventilation and diffusion, while in expiration by trapping of air and obstruction of alveoli it increases vitiation. If the bronchial tonus is severe, some alveoli may be completely shut off from gas exchange. The blood passing through these is not aerated and tends to reduce the effectiveness of aeration in other portions of the lungs. If the obstruction persists, factors other than the compromised conducting airway contribute to the alteration of the respiratory pattern. They are secondary to the obstruction to both phases of breathing and exert their action on the extrapulmonary respiratory apparatus. Inspiration becomes extremely active from central discharges which may be maximum. Their intensity,

though influenced largely by hypercapnia, may be affected by somatic reflexes not unlike those of exercise. This activity does not originate in the lungs since experimentally it follows only marked collapse. Inspiration is characteristically short because of prompt inhibition. The abnormal distention initiates over the vagus a continuous stream of afferent inhibitoinspiratory impulses which are augmented by any further inspiratory effort. Head[20] showed that such prompt inhibition by extreme distention was followed by an abnormally long pause occupied by passive expiration. In asthma, however, expiration is active throughout this period since the air must be expelled through a markedly constricted conducting system. This excitation of expiration is a part of the preceding inhibition by impulses which have sufficient intensity to spread out as excitoexpiratory stimuli. If all these conditions progress, carbon dioxide and oxygen may reach levels in the blood at which carotid sinus or similar reflexes must supply the drive to respiration in the presence of failing central neurons.

Breathing assumes a different form if asthma has produced permanent structural changes in the lungs. Rupture of the septa in emphysema, the common end result of asthma, alters the pulmonary reflexes. Inspiration is almost continuously stimulated by impulses excited in the pressure receptors by the collapse of the septa. The inadequacy of stretch receptor stimulation in the collapsed septa and the slight expansion of the lungs inhibit inspiration poorly. Asthma becomes fixed, much as hypertension, when acute attacks aggravate the compromised respiratory system. Other predominantly vagal effects, similar to the reaction of bronchial muscle, may contribute to obstruction. Mucosal edema, vascular congestion, and excessive secretion commonly occur in clinical asthma, but changes in pulmonary circulation do not appear to be prominent primary factors.

THE CLINICAL TYPES OF ASTHMA

This concept of asthma is supported by the common division of this syndrome into clinical types. In one, definite allergic sensitivity is exhibited in various degrees. The acute rhinitis of early hay fever displays an increased sneeze reflex, but in more severe cases minimal wheezing or typical asthmatic breathing may occur. In the larger portion of this group, breathing is obstructed principally by mucosal edema, congestion, and increased secretion. It is worth noting here that sympathetic denervation by upper dorsal ganglionectomy produces similar changes in the nasal mucosa which persists for some time. During asthmatic attacks insufficient sympathetic activity likely produces comparable changes in the bronchi. Any spastic element of obstruction is a reflex from the upper respiratory tract or a direct effect on muscle. The common relief from atropine in early cases indicates that any direct spastic action on sensitized bronchial muscle is relatively unimportant.

The ability of these mucosal reactions to produce obstruction is clearly illustrated by the clinical features of angioneurotic edema.

The other large asthmatic group manifests no distinct allergy but is usually associated with recurrent infection in some portion of the respiratory tract. The irritation from this infection stimulates the receptors which initiate the reflexes of constriction afferent in the sympathetics and efferent in the vagus. The bronchus is not always the point of origin of this reflex. Sinusitis or other forms of upper respiratory infection which commonly recur in the presence of abnormalities frequently precipitate such reflexes. However, intrapulmonary infection usually initiates this reflex which is always efferent in the vagus. Any direct muscle sensitization must be minimal since skin tests are usually not markedly positive. In all these reflexes the sensory receptors are either sensitized or overstimulated, or the central threshold is lowered. Although the severity and persistence of attacks indicate that spasm is the most important factor, edema, congestion, and increased secretion certainly are present always.

Numerous pharmacologic studies demonstrate the essential hyperactivity of the autonomic nervous system in asthma. Atropine and scopolamine dilate bronchi and reduce secretion by paralysis of the motor parasympathetic endings. Dixon[28] found that pilocarpine and eserine constricted bronchi and increased secretion by stimulating these endings. Sollman and Gilbert[29] observed that the constriction from parasympathetic stimulants was blocked by atropine. Adrenalin dilates bronchi by stimulation of the motor sympathetic endings which inhibit constriction. Any effect upon the mucosal changes depends upon bronchial vasomotor action. Studies on bronchiolar response to anesthetic agents and related drugs yield similar data.[30] Ether, chloroform, and vinethene, which relax bronchi, have been shown to stimulate the sympathetics.[31] Ethylene and nitrous oxide have no significant effects. Cyclopropane produces constriction. In all instances the constrictor response was enhanced by eserine and prevented by atropinization. The frequent occurrence of lower respiratory tract obstruction during cyclopropane anesthesia in patients with asthma or hay fever indicates the hyperactivity of the motor parasympathetic system, which is readily reversed by intravenous atropine or by changing to a sympathetic dilating agent such as ether.[32] The barbiturates have a bronchoconstrictor action.[33]

THE PRINCIPLE OF THERAPY

The rationale of the surgical treatment of asthma rests upon these concepts. In that large group of so-called infective bronchial asthmatic subjects having few allergic manifestations and little benefit from desensitization, surgery has been most useful. Since the spastic factor in these cases involves a reflex originating in the bronchi, which is afferent over sympathetic and efferent over vagal paths, sympathectomy has been

advocated. The afferent path over the stellate and upper thoracic sympathetic ganglia has been interrupted by block or excision in a large number of patients, and the results form the basis of this report. The effectiveness of operation depends upon complete interruption of the afferent portion of the reflex of constriction. Although Leriche and Fontaine[34] and others[35, 36] have observed relief from stellectomy alone, the sensory pathways from one lung are more effectively eliminated by additional section of the upper thoracic sympathetic ganglia. Such wide resection abolishes at least one-half of those afferent impulses from the lungs, which in the presence of a lowered central threshold produce constriction. It does not disturb the normal afferent impulses in the vagus. Interruption of the dilator motor sympathetics by this procedure is evidently unimportant since their activity before operation has not inhibited attacks with maximal constriction. The failures of this method of treatment have been largely in patients whose long-continued asthma has caused emphysema. Three patients subjected to simple unilateral procaine block had immediate onset of status asthmaticus. Previously cited work indicates that this procedure removed a large part of the inhibition of reflex constriction which in these patients is both afferent and efferent in the vagus.

In the allergic group desensitization is the method of choice. It is not unlikely that mucosal changes and possibly direct action on muscle play major roles in the obstruction. If the constrictive element appears prominent but dependent upon a reflex of extrabronchial origin related to infection, the local condition should be corrected. This may indicate the removal of abnormalities such as polyps or treatment of sinusitis or other foci of irritation which initiate the reflexes. Although the afferent limb of these constrictor pathways varies, the efferent is always in the vagus. If this therapy does not obliterate obstruction, the posterior pulmonary plexus resection advocated by Rienhoff[37] may offer the only method of relief. However, this procedure disturbs reflexes which may be essential to normal respiration.

Each patient has been studied extensively before acceptance for surgical treatment. All patients had required frequent hospitalization for intractable asthma which had failed to respond to any and all treatment. Severe bronchospasm after diagnostic lipiodal installations demonstrated little more than extreme sensitivity of the bronchial system. Preoperative bronchoscopy revealed typically allergic mucosa. In every case bronchial irritation was considered the origin of reflex constriction. Through functional respiratory studies, the degree of pulmonary lability has been determined by observing the effects of bronchodilator drugs and nerve block on the vital and breathing capacities and residual air. The observed improvement is an acceptable index of the result that can be expected from interruption. Using these criteria, the following experience with nerve block and ganglionectomy is reported.

NERVE BLOCK

Interruption of sympathetic pathways from the lung by nerve block has been found most useful as a diagnostic and prognostic procedure in the therapy of asthma. By no other method can one be certain that the reflex producing bronchospasm originates in the lungs and traverses the afferent pathway to be interrupted. If this origin cannot be conclusively established, no basis for neurolytic or surgical interruption of the upper dorsal sympathetic pathway exists. Asthmatic patients require special preparations for this procedure. Barbiturates are not used because of the bronchoconstrictor effect. Morphine which may raise dangerously the threshold for respiratory center stimulation is avoided. Scopolamine in doses which do not cloud the effect of block may be used. Thoracic sympathetic nerve blocks are completed with the patient in the sitting position to facilitate respiration. The posterior approach for stellate block and the standard technique of Labat for the thoracic ganglia is recommended. An effective nerve block in this region is indicated by the presence of a homolateral Horner's syndrome and temperature increase with anhidrosis in the affected area. Five cubic centimeters of 1½ per cent procaine solution in normal saline are injected at each ganglion. After ten minutes, if the block is effective with recession of symptoms, 3 c.c. of absolute alcohol are deposited. It should be emphasized that successful sympathetic block may precipitate status asthmaticus in a small minority of patients. Interruption of the motor sympathetic dilator pathways in these patients leaves the motor vagovagal pathway completely uninhibited. Alcohol injections are never to be completed under these circumstances. Therapeutic procedures such as endotracheal intubation to provide oxygen or intravenous atropine may be needed to sustain these patients for the duration of the procaine anesthesia. The stellate is blocked shortly before the thoracic ganglia, since the majority of sympathetic pulmonary pathways pass through it. A favorable block is often dramatic. The patient experiences the sensation of easier breathing and respirations tend to become more normal. Coughing and copious expectoration may follow after several minutes. Most patients complain of dizziness and become pale. Syncope, which is not uncommon, is attributed to dilatation of cerebral vessels. Such dilatation has been observed after experimental stellate block with procaine while the dura was open during intracranial operation.

Pneumothorax, which is a risk in any thoracic nerve block, is a potentially fatal hazard in the asthmatic patient because it is usually tension in type. For this reason nerve block in the status stage is not advocated. It should be completed during a milder period. If it is used in the emergency therapy of status, as has been done in a few cases, the risk of this practice must be recognized.

Ninety-five anesthetic or neurolytic injections of various types have been completed for seventy-two patients. Twenty-four bilateral stellate

blocks were completed for seventeen patients, fourteen of whom had satisfactory sympathetic paralysis. Twelve had definite improvement. Bilateral stellate block was discontinued after a death during the procedure was experienced. This might be explained by the observation of Cromer and Ivy[38] who have reported that bilateral stellectomy sensitized the respiratory center to the impulses rising in the vagus nerve and caused respiratory paralysis. Twenty-eight unilateral stellate blocks have been completed for twenty-five patients. The right was satisfactorily infiltrated for eighteen patients and the left for two. Sixteen of the twenty patients were improved. The right stellate, second and third thoracic sympathetic ganglia were blocked for eighteen patients. Fifteen were improved. The right stellate and second, third, and fourth thoracic ganglia have been blocked for four patients and for two others the fifth was included. Four of these 6 patients successfully blocked were improved. The present practice is to block the afferent sympathetic pathway at the stellate and second and third thoracic ganglia on the right side.

In the entire group of sixty-one patients whose afferent pulmonary sympathetics were successfully blocked, immediate improvement was noted in forty-seven (78 per cent). Although many of these patients could not be followed for an extended time, complete relief has been observed in some for a maximum of thirteen months while in others symptoms have returned in from two to four months. Since nerve block was not effective therapy for prolonged periods, ganglionectomy has been performed. Surgical resection of the stellate and upper thoracic pulmonary sympathetics was limited to patients who had definite benefit from block. One patient in whom stellate block precipitated an acute attack had some relief from procaine block of the vagus in the neck. Then posterior pulmonary plexus resection was completed on one side, but no estimate of the result can be made until the other side is subjected to operation.

OPERATIVE PROCEDURES

Ether given by the carbon dioxide absorption technique with an oral endotracheal airway in place has been utilized for anesthesia. Extrapleural resection of the sympathetics has been attempted. An incision is made between the vertebral border of the scapula and the spine. The muscles are divided and short segments of the upper ribs removed subperiosteally. The ribs are cut as near the spine as possible and preferably removed with some of the transverse process, particularly of the second and third vertebrae. The parietal pleura is mobilized sufficiently to visualize the vertebral artery. The stellate ganglion can then be easily identified with the branches which pass around the artery to the inferior cervical ganglion. After these are clearly identified they are divided as high as possible. The chain is then excised, being careful not to injure the brachial plexus. The excision of the chain is completed

to below the third or fourth ganglion. The lung is then inflated and the wound closed in layers with a catheter in the extrapleural space to prevent trapping of air. It is extremely difficult not to rupture the pleura in mobilizing it. If ruptured, it should be opened widely to prevent tension pneumothorax which is rapidly fatal to these patients.

A persistent Horner's syndrome which developed in all patients did not unduly hinder them. It has gradually improved but never cleared completely. On the operated side the nasal mucous membrane becomes succulent with a marked increase in secretion, mild dilatation of the blood vessels, and considerable edematous swelling. Whether this is related in any way to the cessation of the symptoms of sinusitis, which some of these patients have noted, is not clear at this time. The reaction to adrenalin, either by injection or topical application, seems to be roughly the same on both sides. Skin temperature rises (10 to 12° F.) have been observed invariably on the operated side. This has persisted for more than eighteen months in some cases. All except two patients have had unilateral sympathectomy which is usually sufficient to obtain a good result.

Twenty-one patients have been subjected to operation. The immediate relief previously observed from nerve block was increased by surgical section since there is more effective interruption of the afferent pathways. All patients exhibited additional gradual improvement, probably from the delayed clearing of the lungs, aeration of more alveoli, and possibly adrenalin sensitization. They have been observed for a minimum of three months and a maximum of two years. All have had complete relief of asthma since operation. None have needed bronchodilator drugs despite the fact that most of the patients have not known where "to stick the next shot" since they were so sore from almost constant medication. This is not unexpected since only patients having good response to block have been submitted to operation.

A detailed report of the techniques for nerve block and surgery together with a complete account of the results obtained is in preparation.

SUMMARY

1. The breathing in some types of asthma is the response to exaggerated reflex activity in pathways that carry normal respiratory reflexes.

2. A review of the pertinent anatomic and physiologic evidence is cited to construct these reflex pathway patterns. The excursion in the respiratory-conducting airway system essential to normal breathing involves marked relaxation and constriction of the bronchial muscles which may not be reflex in type.

3. Impulses, reflex in type, that produce both constriction and dilatation of the bronchi have been clearly demonstrated in these pathways.

The reflex of constriction is afferent with the sympathetic division of the autonomic nervous system and efferent in the vagus nerve. The reflex of dilatation is afferent in the vagus and efferent in the sympathetics. It is inhibitory in type.

4. The reflex afferent impulses of constriction travel predominantly over fibers in sympathetic pathways which may be interrupted at the stellate and second, third, and fourth thoracic ganglia.

5. Bronchial asthma precipiated by recurrent infection in the bronchi is dependent, not unlikely, upon excitation of this reflex of constriction. It can be benefited by interrupting this reflex before damage to the lungs has become irreversible.

6. Blocking the pathway of this reflex is the most reliable method for determining its pulmonary origin. If such origin is not demonstrated, no indication for interruption of the pathway exists. Furthermore, nerve blocking is useful to differentiate the types of asthma.

7. The application of neither anesthetic nor neurolytic solutions has resulted in permanent interruption of these pathways. The results from surgical excision have been more complete and permanent.

8. The experience of this study demonstrates that surgery is an important merited therapeutic procedure if it is restricted to patients with asthma of pulmonary origin who have been carefully selected after benefit from nerve blocking.

REFERENCES

1. Lumsden, T.: Observations on the Respiratory Centers, J. Physiol. **57**: 354-367, 1923.
2. Stella, G.: On the Mechanism of Production and Physiological Significance of Apneusis, J. Physiol. **93**: 10-23, 1938.
3. Bronk, D. W., and Ferguson, L. K.: The Nervous Control of Intercostal Respiration, Am. J. Physiol. **110**: 700-707, 1935.
4. Pitts, R. F., Magoun, H. W., and Ranson, S. W.: The Origin of Respiratory Rhythmicity, Am. J. Physiol. **127**: 654-670, 1939.
5. Ranson, S. W., and Billingsley, P. R.: An Experimental Analysis of the Sympathetic Trunk and Greater Splanchnic Nerve in the Cat, J. Comp. Neurol. **29**: 441-458, 1918.
6. Chase, M. R., and Ranson, S. W.: The Structure of the Roots, Trunk, and Branches of the Vagus Nerve, J. Comp. Neurol. **24**: 31-60, 1914.
7. Larsell, O., and Mason, M. I.: Experimental Degeneration of the Vagus Nerve and Its Relations to the Nerve Terminations of the Lung of the Rabbit, J. Comp. Neurol. **33**: 509-516, 1921.
8. Head, H.: On the Disturbances of Sensation, With Especial Reference to the Pain of Visceral Disease, Brain **16**: 1-133, 1893.
9. Rossi, Ottorino: On the Afferent Paths of the Sympathetic Nervous System With Special Reference to the Nerve Cells of the Spinal Ganglia Sending Their Peripheral Processes in the Rami Communicantes, J. Comp. Neurol. **34**: 493-505, 1922.
10. Ranson, S. W.: Afferent Pathways for Visceral Reflexes, Physiol. Rev. **1**: 447-522, 1921.
11. Ranson, S. W., and Billingsley, P. R.: The Superior Cervical Ganglion and the Cervical Portion of the Sympathetic Trunk, J. Comp. Neurol. **29**: 313-358, 1918.
12. Ranson, S. W., and Billingsley, P. R.: The Thoracic Truncus Sympatheticus, Rami Communicantes and Splanchnic Nerves in the Cat, J. Comp. Neurol. **29**: 405-439, 1918.
13. Möllgaard, H.: Studien über das respirationische Nervensystem bei den Wirbelthiere, Skandin. Arch. f. Physiol. **26**: 315-383, 1912.

14. Dixon, W. E., and Brodie, T. G.: Contributions to the Physiology of the Lungs: Part 1, The Bronchial Muscles, Their Innervation and the Action of Drugs Upon Them, J. Physiol. **29**: 97-173, 1903.
15. Dixon, W. E., and Ranson, F.: Broncho-dilator Nerve, J. Physiol. **45**: 413-428, 1912.
16. Rasmussen, A. T.: Pathways for Nervous Reflexes From the Parenchyma of the Lung, Am. Rev. Tuberc. **13**: 545-549, 1926.
17. Larsell, O.: Nerve Terminations in the Lung of the Rabbit, J. Comp. Neurol. **33**: 105-131, 1921.
18. Ploschko, A.: Nervendigungen und den Ganglien der Respirationsorgane, Anat. Anz. **13**: 12-22, 1897.
19. Carlson, A. J., and Luckhardt, A. B.: Lung Automatisms and Reflexes, Am. J. Physiol. **55**: 122-137, 1921.
20. Head, H.: On the Regulation of Respiration, J. Physiol. **10**: 1-70, 1889.
21. Gault, C. C., and Scott, F. H.: Some Points in the Nervous Relation of Respiration in the Cat, Am. J. Physiol. **45**: 554-566, 1918.
22. Coombs, Helen C.: The Relation of the Dorsal Roots of the Spinal Nerves and the Mesencephalon to the Control of Respiratory Movements, Am. J. Physiol. **46**: 459-471, 1918.
23. Combs, Helen C., and Pike, F. J.: The Role of Afferent Impulses in the Control of Respiratory Movements, Am. J. Physiol. **45**: 569-581, 1918.
24. Barry, D. T.: Apparent Impressions From the Respiratory Mechanism, J. Physiol. **45**: 473-481, 1912.
25. Larsell, O., and Burget, G. E.: The Effects of Mechanical and Chemical Stimulation of the Tracheal Bronchial Mucous Membrane, Am. J. Physiol. **70**: 311-321, 1924.
26. Craigie, E. H.: The Reflex Produced by Chemical Stimulation of the Deeper Respiratory Passages, Am. J. Physiol. **59**: 346-352, 1922.
27. Macklin, Charles C.: Musculature of the Bronchi and Lungs, Physiol. Rev. **9**: 1-60, 1929.
28. Dixon, W. E.: The Production of Bronchial Spasm, M. J. & Rec. **130**: 576-582, 1929.
29. Sollman, I., and Gilbert, A. J.: Microscopic Observations of Bronchiolar Reactions, J. Pharmacol. & Exper. Therap. **61**: 272-285, 1937.
30. Adriani, J., and Rovenstine, E. A.: Autonomic Response of Bronchial Tissue to Various Anesthetic Drugs, Proc. Am. Physiol. Soc., p. 4, 1941.
31. Knoefel, P. K.: Anesthesia and the Sympathetic Nervous System, Anesth. & Analg. **15**: 344-350, 1936.
32. Phelps, McKinnie L., and Rovenstine, E. A.: Respiratory Obstruction During Cyclopropane Anesthesia from Bronchial Constriction, J. THORACIC SURG. **11**: 565-570, 1942.
33. Burstein, C. L., and Rovenstine, E. A.: Respiratory Parasympathetic Action of Some Shorter Acting Barbituric Acid Derivatives, J. Pharmacol. & Exper. Therap. **63**: 42-50, 1938.
34. Leriche, Rene, et Fontaine, Rene: Resultats Eloignes du traitement de L'Asthme Bronchique par la Stellectomie, Presse med. **47**: 241-243, 1939.
35. Phillips, E. W., and Scott, W. J. Merle: The Surgical Treatment of Bronchial Asthma, Arch. Surg. **19**: 1425-1456, 1929.
36. Galup, J.: Non-paroxysmal Dyspnea a Desequilibra Neurovegatatif, Presse med. **36**: 686-688, 1939.
37. Rienhoff, Jr., W. F., and Gay, Leslie N.: Treatment of Intractable Bronchial Asthma by Bilateral Resection of Posterior Pulmonary Plexus, Arch. Surg. **37**: 456-469, 1938.
38. Cromer, S. P., and Ivy, A. C.: Respiratory Death from Central Vagus Stimulation After Removal of Stellate Ganglia, Am. J. Physiol. **104**: 457-475, 1933.

FASCIA LATA TRANSPLANT FOR DIFFICULT HERNIAS

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WE ARE all cognizant of the fact that in the evolution of the surgical art—as of other arts, plastic or graphic—there were epochal discoveries and innovations which served as symbolic milestones. This is particularly true of the progress of hernial surgery, but with the important reservation that the aforesaid milestones were few indeed on the horizon-less road of history. For over 800 years there was a Dark Age in hernial surgery. Primitive concepts held sway to within comparatively recent years concerning the etiology of this world-wide affliction, concepts, and practices which to us moderns, would seem to transcend the limits of human credulity but which received the sanction of "surgical" custom.

I do not refer merely to the ancient surgical customs, for be it remembered that it was not until Joseph Pancoast's day—in the 1860's—that any one in the United States attempted to operate on reducible inguinal hernia, such was the great dread and near certainty of a fatal issue.

Bassini of Padua described novel operations for inguinal and femoral hernia which proved revolutionary. They were, in effect, the first rational advances in the surgical repair of hernia in over 2,000 years, since Babylonian and ancient Egyptian days. Bassini's operation was based on a sound anatomic basis and therefore received world-wide acceptance.

W. S. Halsted almost concurrently with Bassini devised a new operation for inguinal hernia which in some regards closely resembled that of Bassini, in which an attempt was made to re-establish the obliquity of the canal. The spermatic cord was placed on the internal oblique muscle. Halsted constructed a new canal and ring but transplanted the spermatic cord superficial to the aponeurosis of the external oblique muscle.

A number of other innovators in surgery advocated the use of various flaps of fascia, periosteum or bone in one way or another to overcome the admittedly unsatisfactory results in hernial surgery. Kirschner, with this objective in view, in 1910, first suggested the feasibility (and later recanted somewhat) of repairing various anatomic defects with a sheet or strips of fascia lata. In the same year Mackenzie utilized the iliotibial band of the tensor fasciae latae in the repair of large abdominal wall defects.

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McArthur, in 1901, was the first to employ living fascia as suture material. He utilized a narrow pedicled strip from the aponeurosis of either flap of the split external oblique and sutured the structures to the aponeurosis with these strips.

Gallie and LeMesurier's great contributions to hernial surgery are, of course, well known. They demonstrated, with clinical and experimental evidence derived from rabbits and dogs, that long strips of fascia lata were ideal suture material for the repair of otherwise inoperable or recurrent hernias (Figs. 1 and 2).

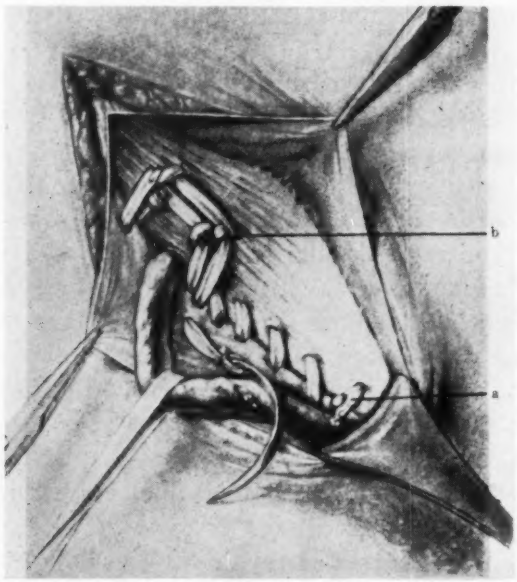

Fig. 1.—Repair of inguinal hernia with sutures of fascia lata. Insertion of first row of sutures. (*a*) Slipknot anchoring stitch. (*b*) Lock stitch. (Courtesy of Dr. W. E. Gallie.)

Gallie and LeMesurier's results found considerable confirmation in the later researches of Gratz[1] concerning the tensile strength and elasticity of human fascia lata. This investigator found that, "The material showed surprisingly great tensile strength, comparing favorably with soft steel wire of the same weight; in addition, it showed an unexpected degree of elasticity."

While many surgeons today (Seelig and Chouke[2]) have serious doubts concerning the value of ordinary sutures in fixing the conjoined muscles and the inguinal ligament they are not, on the other hand, in agreement with the Gallie and LeMesurier contention that no postoperative evidence is found of previous fixation with the usual technique. It is contended that the Canadian surgeons failed in their dissections to identify properly the atrophic, frayed-out muscles—often all that remains following fixation. Muscle, deprived of function, rapidly atrophies; this change is hastened by the constant pressure to which it is subjected. This holds particularly true, it is maintained,

when the transversalis fascia is defective and there is a concentration of intra-abdominal pressure perpendicular to its fibers. Careful observation will demonstrate, at the least, the margin of the muscle still adherent to the ligaments, though the fibers may be so separated and frayed that recurrence is not surprising.

Seelig and Chouke[2] undertook to investigate the moot question of the union of muscle and fascia. They concluded that "normal muscle will not unite firmly with fascia and ligament. It is, therefore, a useless procedure to suture the abdominal muscles to the inguinal ligament in the hope of buttressing a weak or ruptured abdominal wall."

A. R. Koontz,[3] on the other hand, demonstrated clearly that muscle united with fascia, owing to the union of the latter with the fibrous components of the former.

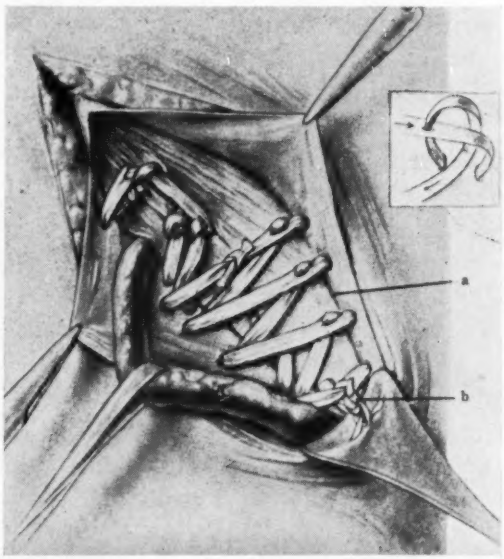

Fig. 2.—Repair of inguinal hernia. Insertion of second series of sutures between external aponeurosis and inguinal ligament. (*a*) Junction line of aponeuroses of internal and external oblique muscles. (*b*) Termination of suture. Insert: practical lock stitch. (Courtesy of Dr. W. E. Gallie.)

Many surgeons object to the use of autogenous fascial sutures in hernial surgery. It is argued that infections are more common than with the older methods and that the introduction of large needles through the transversalis fascia and the inguinal ligament frequently leaves weak spots which predispose to recurrences. Burdick and associates[4] came to the conclusion, after 1,485 hernia operations, that the theory of fascial sutures for repair is based on an erroneous surgical principle!

Some surgeons, further dissenting, state that the use of the iliotibial tract of the tensor fasciae femoris muscle is unsound in surgical principle and wholly devoid of the merits commonly ascribed to the technique for the procurement of transplants therefrom. The rejoinder,

nevertheless, from the proponents of the ingenious surgical procedure, is that it is justified on the following theoretic and practical grounds.

1. It is, above all, an attempt to produce better results.

2. Closure of large defects in the floor of the inguinal canal is not invariably successful.

3. Strips of fascia are nonabsorbable and do not tear through after insertion.

4. It is evident that living fascial sutures, unlike other sutures, do not drag the inguinal ligament and the tendo conjunctus together, distorting their relationships. The integrity of the inguinal ligament is preserved with their use. The darning serves as a re-enforcement in Hesselbach's triangle.

5. The iliotibial band is the only accessible and mobilizable fascial musculature available, retaining its myodynamic integrity, that is, elasticity, contractility, and extensibility. The viability and therefore the tensile strength of a graft depends, first, on its connection with a base and the preservation of its nerve and vascular supply and second, on the width of the link. The nerve and vascular supply, highly situated, are not disturbed or destroyed.

6. The requisite amount of fascia is procurable from both thighs, if necessary.

7. There is no loss of bodily function in the transference of the graft.

8. The contiguous biceps femoris and vastus lateralis muscles do not herniate much, if at all, because they have their own fascial enveloping sheaths.

9. Surgical experience sustains the validity of the foregoing statements. The transplantation of a live structure with intact nerve and blood supply is fully effective because the tonus of this musculofascial structure is preserved. An abdominal wall defect becomes such postoperatively by virtue of the fact that the tissues utilized to close it have no tonus. The collagen fibrils which constitute fascia possess the sole function of constant tension in an elastic manner; the requisite tonus is thus maintained. An examination of the groin in a patient for whom a fascial transplant has been utilized reveals the firmness, and it may be added, the contractility and tonicity, of the band. In brief, instead of closing a physiologic anatomic defect with a lifeless structure, a viable physiologic and anatomic one is used.

The strongest union takes place when white fibrous tissues are approximated without tension. When two living fascial planes are approximated there is complete and enduring union.

It is manifest, therefore, that the use of living fascia in the repair of large hernias is of transcendent value as compared to all other forms of suture material.

The common experience of surgeons has been that grafts of animal tissues, unless they are autogenous, are unsuccessful, being fated to absorption and other changes.

Heterogenous fascial sutures, it is stated, function by stimulating tissue repair, but I have not observed any advantage in their use. Contrariwise, these foreign structures often undergo necrosis and degeneration. The employment of heterogenous fascial sutures, it seems to me, should be discouraged.

Anatomists and surgeons have for a long time been aware of the fact that fascial and aponeurotic structures are the main supporting and enveloping tissues of the body. Surgeons utilized fasciae, aponeuroses and their derivatives, to obliterate defects (congenital, acquired postoperative, and traumatic) in the abdominal wall and inguinal regions. An approach to perfection in the use of aponeuroses and fasciae has not perhaps been thought possible because of our failure to take advantage of what one may call the "natural resources" of the human body.

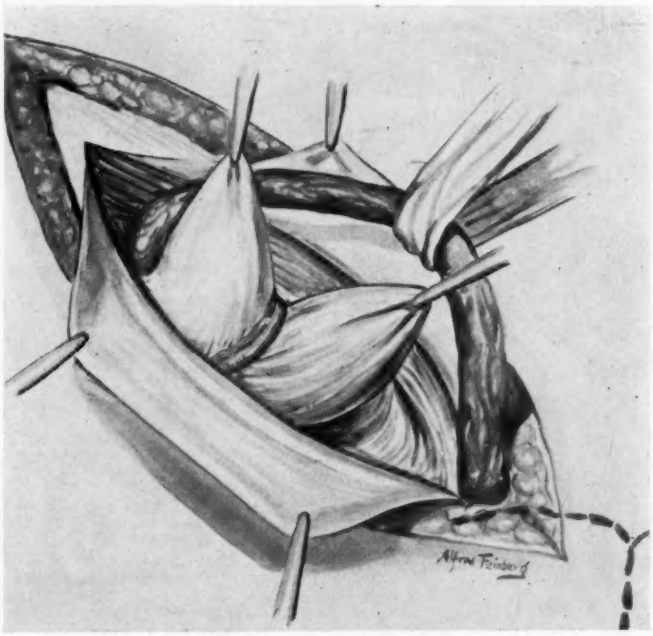

Fig. 3.—Pantaloon or saddlebag hernia; inferior epigastric vessels forming a bilocular sac. Condition found in Case 2.

Architecturally, the inguinal canal may be considered a funnel-shaped structure. From a purely mechanical viewpoint it is, therefore, sound surgery to obturate the inlet to this funnel rather than the outlet in order to prevent effectively the intrusion of an abdominal viscus, part thereof, or of an associated structure.

One does not marvel at the nearly universal, excellent results obtained by the orthopedists in tendon transplants, to wit, the utilization of the peroneus longus in paralysis of the tibialis anterior muscle, and the role played by the teres major muscle in its transplantation in

Erb's birth palsy. The success of the procedure is undoubtedly dependent on the fact that living structures are used.

The paramount advantages of the utilization of fascia lata transplants, stated succinctly, are as follows.

1. Fascia lata transplant has a definite role in the repair of abdominal wall defects.

2. In direct and pantaloon hernias, with the high incidence of recurrence, it rarely fails (Figs. 3 and 4).

3. In hernias of the older group, of men and women past 50 years of age, it promises cures.

4. In the repair of "sliding hernias" (a problem in the past) a solution is at hand.

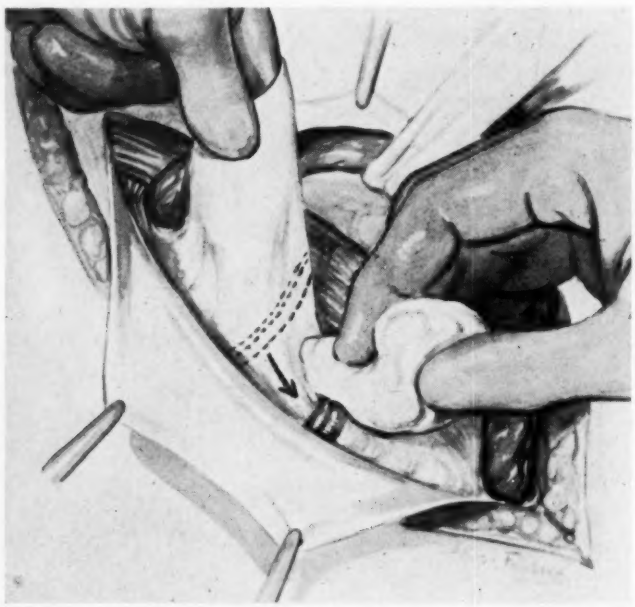

Fig. 4.—Pantaloon hernia converted into an indirect inguinal hernia by making traction on indirect portion and dislocating inferior epigastric vessels medially.

5. Despite wound infection, the results are excellent.

6. No disturbances of locomotion ensue.

7. Recurrences are rare.

Stated briefly, the indications for the utilization of fascia lata transplants are:

1. Hernias difficult to repair by the customary surgical methods.

2. Large or massive inguinal hernias, complete or incomplete, direct or indirect, with a structural defect in Hesselbach's triangle, and musculature and aponeuroses which have lost their myodynamic integrity.

3. Recurrent hernias with large defects in Hesselbach's triangle (Fig. 5).

4. Large or massive incisional hernias (Fig. 6).

The quintessence of the matter lies in the following: When extreme relaxation of the inguinal canal floor is present with loss of support in the inguinal triangle consequent upon the inadequacy of the transversalis fascia from one cause or another, whether it be weakness or absence, with loss of valvular function of the rings and loss of the physiologico-anatomic functions of the abdominal wall bulwark, the utilization of fascia lata transplants in a deep reconstructive repair has unparalleled values.

Fig. 5*A* (Case 5).—Right recurrent inguinal incisional hernia, and left saddlebag hernia.

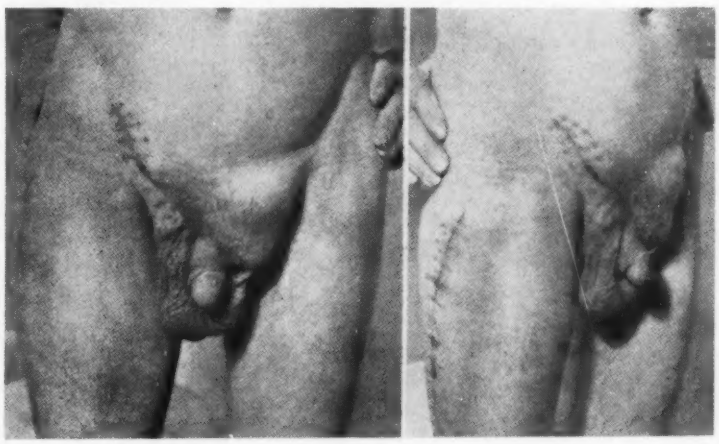

Fig. 5*B* (Case 5).—After pedicle fascia lata transplant for right recurrent inguinal incisional hernia.

Fascia lata transplants, or any other variety of transplants, should not be used indiscriminately, whether in the groin or elsewhere in the body. Where the fascia transversalis has not lost its prime function, it is, in my opinion, an unsound surgical procedure. As in all ameliorative efforts, medical or surgical, patients should be strictly individualized.

In 1934, Wangensteen[5] described his method of repairing inguinal hernia by means of a pedicled graft from the iliotibial tract. The flap

is swung into the wound over the inguinal ligament and sutured over the spermatic cord.

Certain modifications of surgical technique were found advisable (Fig. 7).

Fig. 6 (Case 6).—Woman, aged 40 years, with large postoperative incisional hernia.

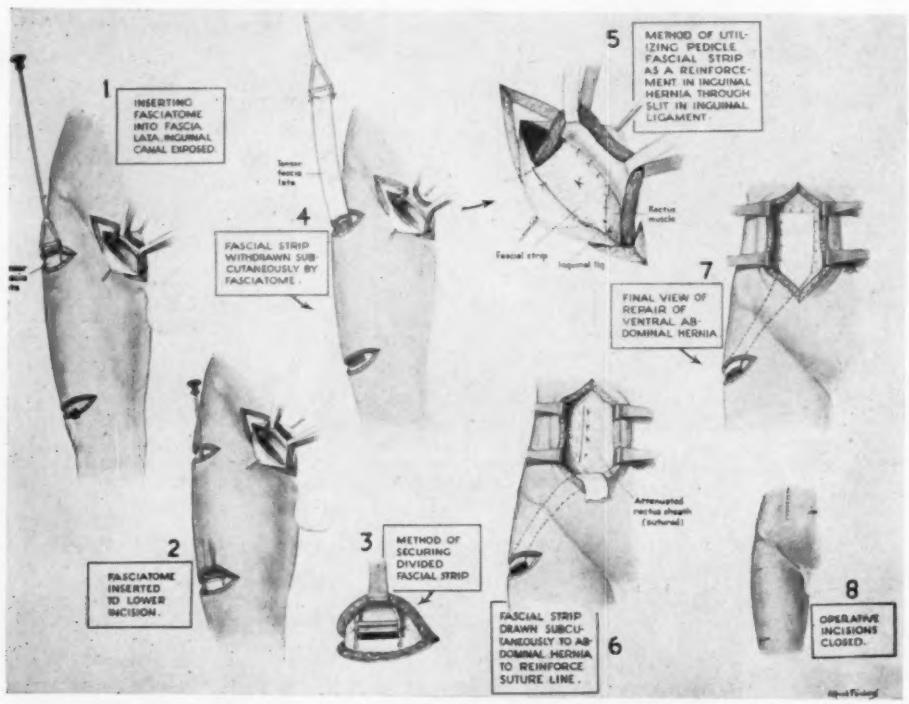
1
INSERTING FASCIATOME INTO FASCIA LATA. INGUINAL CANAL EXPOSED.
2
FASCIATOME INSERTED TO LOWER INCISION.
3
METHOD OF SECURING DIVIDED FASCIAL STRIP
4
FASCIAL STRIP WITHDRAWN SUBCUTANEOUSLY BY FASCIATOME.
5
METHOD OF UTILIZING PEDICLE FASCIAL STRIP AS A REINFORCEMENT IN INGUINAL HERNIA THROUGH SLIT IN INGUINAL LIGAMENT.
6
FASCIAL STRIP DRAWN SUBCUTANEOUSLY TO ABDOMINAL HERNIA TO REINFORCE SUTURE LINE.
7
FINAL VIEW OF REPAIR OF VENTRAL ABDOMINAL HERNIA
8
OPERATIVE INCISIONS CLOSED.

Fig. 7.—Pedicle fascia lata transplant in inguinal and ventral hernias.

I utilize the following technique for fascial sutures, a plastic operation for difficult inguinal hernias. The usual inguinal incision is made and the external oblique aponeurosis is split parallel to the inguinal canal. Extreme care must be taken to identify the external iliac,

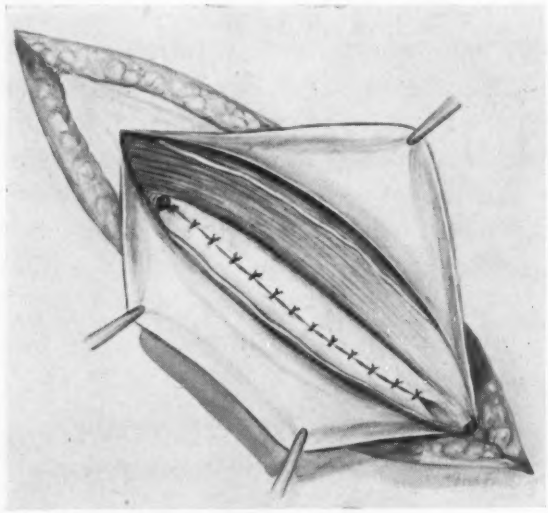

Fig. 8.—Redundancy of transversalis fascia in Hesselbach's triangle eliminated by interrupted plicating sutures.

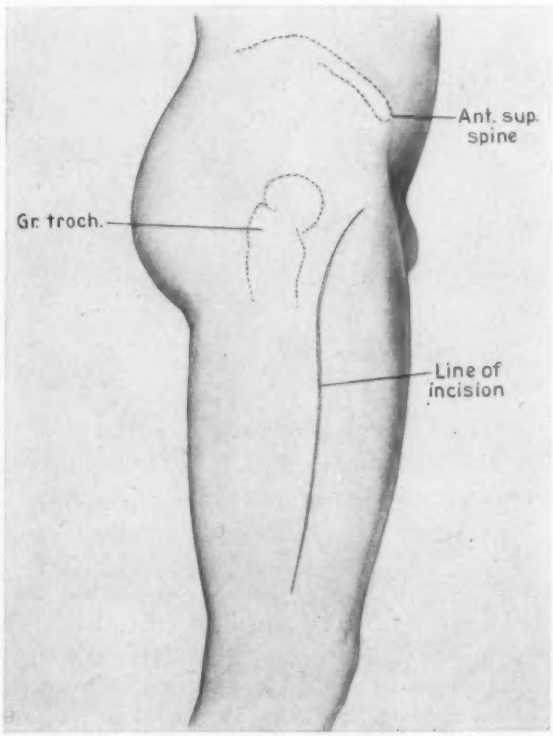

Fig. 9.—Skin incision for fascia lata transplant.

femoral and inferior epigastric vessels, in inguinal hernia. The external oblique aponeurosis is then divided parallel to the inguinal canal to a point just beyond the abdominal inguinal ring, being careful to avoid injury to the subjacent ilio-inguinal nerve. When there is a marked redundancy of the transversalis fascia in Hesselbach's triangle, it is taken up by a series of interrupted sutures inserted similar to vertical Lembert sutures (Fig. 8). A vertical incision is then made along the anterolateral aspect of the thigh, its upper end curved somewhat anteriorly from a point above the anterior superior angle of the great trochanter, downward as far as required, revealing the iliotibial band (Fig. 9). A sufficient section of this band is taken and of requisite width to reach, without tension, the entire hernial defect (Figs. 10 and 11).

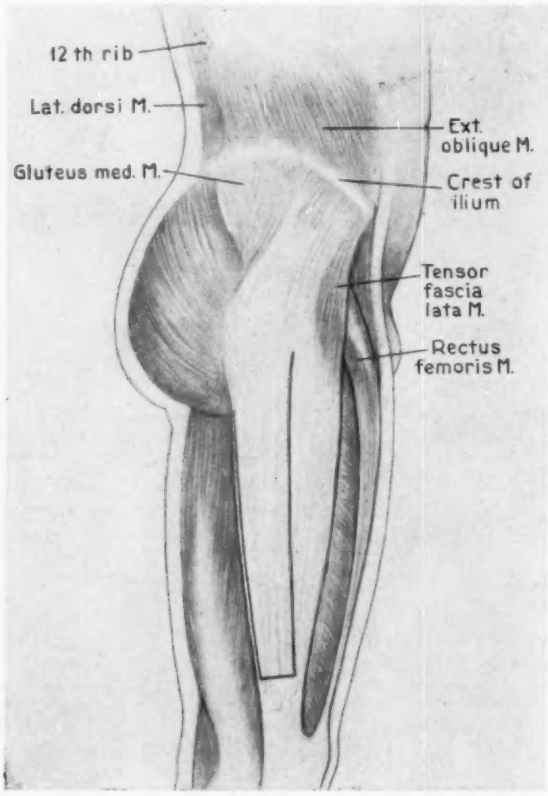

Fig. 10.—Delineation of the strip of tensor fascia lata for fascial transplant.

A strip of fascia, three inches wide and two to four inches long, is usually sufficient, so that any tendency to herniation of the vastus lateralis muscle is averted. The pedicled fascial strip is then carried to the inguinal ligament subcutaneously. A slit is made in the inguinal ligament through which the fascial transplant is drawn. Three sutures are placed at this point in the ligament uniting the medial and lateral boundaries of the slit to the nearest portion of the fascial trans-

plant and the center of the inguinal ligament (close to the slit) to the middle of the transplant. Three sutures are inserted, one at each angle of the gash and one in the center of the inguinal ligament. The free end of the transplant is then sutured to the tendo conjunctus and to the inferior aspect of the external oblique aponeurosis by No. 1 interrupted chromic catgut strands placed at intervals of about three-quarters of an inch. Care must be observed that the medial angle of the free edge of the pedicle is sutured sufficiently far medially so that it impinges upon the rectus abdominis muscle; the lateral edge of the free border of the fascial transplant must be sutured far enough to impinge upon the deep inguinal ring. The medial border of the fascial transplant above the inguinal ligament is then sutured to the superior aspect of the lacunar ligament to prevent herniation. A single suture is then placed, approximating the middle section of the transplant above the inguinal ligament to the underlying transversalis fascia in

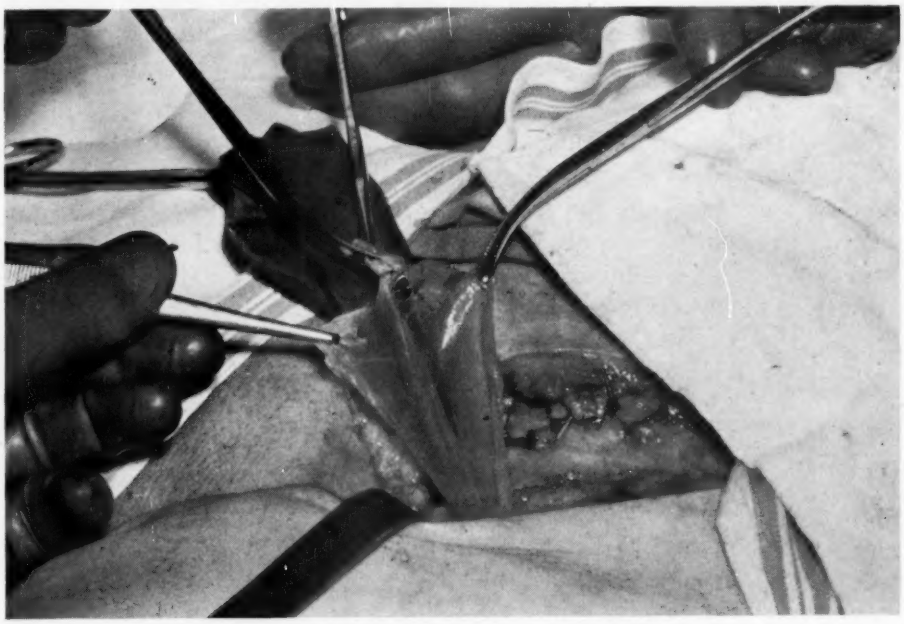

Fig. 11.—Pedicle flap of tensor fascia lata brought up as a re-enforcement subcutaneously.

order to eliminate dead space. The lower free border of the external oblique aponeurosis is finally sutured to the upper free margin (in the manner of the Wyllys Andrews repair) thereby placing the spermatic cord subcutaneously. Thorough hemostasis, as in all plastic surgical procedures, must be carried out. The skin is closed with black silk sutures.

In fashioning the fascial flap it is absolutely essential to obtain one sufficiently long to obviate tension on the suture line. Care must be

taken not to injure the external iliac or femoral vessels in splitting the inguinal ligament. The lateral margin of the flap, in the inguinal region, should be close enough to impinge upon the deep inguinal ring and yet not too far so as to constrict the spermatic cord and its constituents. The medial border of the flap, in the inguinal region, must be firmly secured at its lower margin to the lacunar ligament.

Slight variations in technique are followed (depending upon expediency) in repairing abdominal wall defects with these grafts. As Wangensteen aptly stated, "In the repair of incisional hernias, the flap is best sutured as a patch over the site of the closure, it being anchored securely to the normal adjacent fascial structures."

The fascia lata through the medium of the tensor fasciae femoris muscles has the crest of the ilium as an anchorage. The external oblique aponeurosis to which it is sutured has the lower seven ribs as an anchorage through the medium of the external oblique musculature. Thus, we have two firm structures alive with myodynamic attributes of contractility and mobility pulling against each other in a synergistic manner. Therefore, the best physiologic cure for an abdominal defect is utilized.

Recently I devised a fascial stripper, a fasciatome, enabling one to obtain pedicled fascia flaps of various widths subcutaneously through a small incision in the upper part of the thigh and one just above the knee. Thus the long scar, an objectionable feature of the procedure just described, is eliminated. A subsequent report will demonstrate the use of this instrument.

TABLE I

EIGHTY CASES OF FASCIA LATA TRANSPLANTS

Sex:	
Males	75 cases
Females	5 cases
Age:	
Youngest	25 M
Oldest (sliding hernia)	67 M
Average	48 plus
Types:	
Indirect inguinal	3 cases
Direct inguinal	10 cases
Saddlebag or pantaloon	30 cases
Bilateral saddlebag	1 case
Sliding	11 cases
Incisional inguinal	19 cases
Incisional abdominal	6 cases
Total	80
Recurrences:	
Incisional inguinal	3 cases
Incisional abdominal	1 case
Total	4

There were only four recurrences in my series of eighty cases with the use of fascia lata transplants. A bilateral fascia lata transplantation was done in one patient with a subsequent recurrence on one side, and in two I failed to suture the medial part of the transplant to the lacunar ligament, thus permitting a bulge to occur in that area. In the fourth case, a postoperative incisional hernia, a defect recurred at the upper angle of the wound.

REFERENCES

1. Gratz, C. M.: J. Bone & Joint Surg. **13**: 334-340, 1931.
2. Seelig, M. G., and Chouke, K. S.: Arch. Surg. **7**: 553-572, 1923.
3. Koontz, A. R.: Surg., Gynec. & Obst. **42**: 222-227, 1926.
4. Burdick, C. G., Gillespie, D., and Higinbotham, N.: Ann. Surg. **106**: 333-343, 1937.
5. Wangensteen, O. H.: Surg., Gynec. & Obst. **59**: 766-780, 1934.

FREE AUTOPLASTIC TRANSPLANTS OF FASCIA LATA IN THE REPAIR OF LARGE INCISIONAL HERNIA

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HISTORIC REVIEW

THE anatomic closure of hernial defects described by Bassini and Halsted marked the advent of modern hernial surgery. The failure of these methods in the treatment of many large and difficult hernias had early stimulated surgeons to search for suitable strengthening materials. All methods using nonabsorbable foreign materials, such as celluloid plates, silver screens, paraffin, metal staples, etc., have been abandoned. The almost unanimous experience of surgeons has been that grafts of animal tissues, unless they are autogenous, are unsuccessful, being fated to absorption, to calcification, to mere toleration as of a foreign body, to sloughing, or to replacement with connective tissue.

The major advance in the surgical treatment of hernia has been the utilization of fascia. Wölfler, in 1892, and later Halsted turned down a flap of anterior rectus sheath and sutured it to Poupart's ligament. In 1901, McArthur used as a fascial suture a strip cut from, but left attached to, the adjacent edge of the incised external oblique aponeurosis. Then followed the development of three plastic procedures which are more or less in popular use today. Kirschner first demonstrated, in 1909, the successful use of free autoplastic fascia lata transplants. In 1921, Gallie used "living sutures" of fascia lata strips to fill hernial defects by weaving them into a latticework. Wangensteen, in 1933, employed the iliotibial tract of fascia lata as a pedicled flap in the repair of recurrent and difficult hernias and other large defects of the abdominal wall. The use of free fascial grafts has enjoyed frequent reports in the German literature but has received scant attention by American writers. We believe that this method has not received the acclaim that it deserves and that a review of its background will establish a convincing basis for its use.

Clinical and experimental efforts have been made from time to time in the past fifty years to transplant free grafts of fascia for various purposes, but the first systematic study of the subject was made in 1909, by Kirschner. He found after many animal experiments, in which various layers of the abdominal wall were sacrificed, that he could prevent the development of hernias with free fascial transplants. After a great number of operations on man without a single case of disturbance of healing under aseptic conditions, Kirschner concluded that the heal-

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ing tendency of fascia must be extraordinarily great to explain his large series of successes. He recognized the resistant quality of transplanted fascia even under the unfavorable circumstances of bad nutritional and septic conditions. Ordinarily, the graft would be subject to the cementing action of ingrowing connective tissue on both surfaces, but even when healing could occur on only one surface, such as when the graft faced the free peritoneal cavity, the result was successful. Furthermore, a transplant poorly nourished on both sides, such as one replacing the layers of the abdominal wall including peritoneum, and not covered by skin so that it was visible at every dressing, also remained viable. Dural defects could be grafted successfully even though one side is covered by bone and the other exposed to protein-poor cerebrospinal fluid. He cited one case, out of many, of fascial replacement of an abdominal defect secondary to a huge appendical abscess; this too was successful despite the presence of infection. Transplants under such conditions are not recommended, but such cases illustrate the extraordinary healing tendency of fascia.

FATE OF THE TRANSPLANT

Considerable controversy as to the histologic fate of transplanted fascia still exists. Kirschner observed grafts for 101 days and found that they retained their characteristic structure and healed practically unchanged. Experiments by Dean Lewis and Davis strongly supported this observation. Other observers found some evidences of degeneration, vascularization, and replacement, but Kirschner felt that the question of complete viability as such was, after all, purely academic since partial replacement by scar tissue made the transplant no less serviceable than normal fascia.

Neuhof, in a critical review of Kirschner's work, felt that he did not establish the viability of transplanted fascia by his experiments, but that he did place the method on a sound working basis. A summary of the prevailing thought on the subject is supplied by Neuhof: Fascia is a simple tissue, relatively poor in cells and blood vessels and is one of the most readily transplantable of all tissues, healing with minimal reaction, even under unfavorable circumstances. The early phenomena occurring about an autograft of fascia are agreed upon by all investigators. The transplant is promptly covered by a layer of fibrin, rich in leucocytes, that gradually undergoes organization, so that in two weeks a thick zone of cellular granulation tissue is found to surround the graft. The staining quality of the fascial cells as well as the intercellular substance suffers almost from the outset, indicating some degree of degeneration. Apparent recession of the degenerative phenomena is seen in the succeeding weeks. Is this really the case or are we dealing with the early evidences of gradual replacement? Mimicry of the appearance of any original graft by the replacing tissue occurs frequently. The question of the persistence or replacement of fascial transplants has been

widely discussed. A large part of the experimental studies undertaken to settle this moot point must be ruled out because the periods of observation after transplantation are too brief. Permanent viability cannot be accepted as established since replacement phenomena and evidence of degeneration do exist. Experiments show that the general architecture of the graft is preserved for as long as six months to one year, but careful examination reveals the fact that the transplant is not preserved as such, but has been gradually replaced by fibrous connective tissue closely resembling it. That this replacement is very gradual is evident from the fact that the phenomena are not complete even after one year. This new tissue cannot be termed cicatricial, the resultant of degeneration, but may be more accurately described as a cellular connective tissue occupying the framework and largely maintaining the size and form of the original transplant. As a result, the purpose for which the transplantation has been performed can be ordinarily achieved. The ready transplantability of fascia and its great practical usefulness have been established on a firm basis.

THE FASCIA LATA

The fascia lata forms a uniform investment for the whole of the thigh but varies in thickness in different parts. The portion arising from the front part of the crest of the ilium, corresponding to the origin of the tensor fasciae femoris muscle, passes down the outer side of the thigh as two layers, one superficial to and the other beneath this muscle. At the lower end of the latter, which is about one-quarter of the way down the outer side of the thigh, these layers become blended into a broad thick band, which is continued downward to the knee. This iliotibial tract is the densest portion of the fascia lata and is the most desirable for transplantation. The histology of human fascia closely resembles that of the dog and rabbit, which are the experimental animals commonly used in work on this subject. Throughout most of its extent, it is composed of three layers. The fibers of the middle, the most dense and thickest of the three, run in the vertical axis while the fibers of the inner and outer layers course at right angles to the main median layer. Each of the strata is composed of closely woven bundles of wavy connective tissue, microscopically resembling tendon tissue. The median portion is joined to the inner and outer layers by loose connective tissue, which carries the blood vessels and most of the elastic fibers.

Fascia lata is superior to other similar tissues. It is easily accessible. Throughout its course it lies under the skin and a simple incision effects adequate exposure. It is readily freed from the subcutaneous fat and is easily detached from the underlying muscle, unlike the anterior rectus sheath with its tendinous intersections. The supply is plentiful even for the most extensive autoplasty. Especially is this true when both thighs are available. Successful grafts measuring nine inches square are not unusual. Its sacrifice is harmless. This does not hold when ab-

dominal fascia is used. Occasional herniation of the underlying muscle can be minimized or completely avoided if the margins of the defect in the fascia following excision of the patch are tacked down to the underlying muscle; spreading of the remaining defect is thus prevented. It has great strength combined with limited elasticity. Of the various fasciae in the human body, the fascia lata is the strongest, particularly in the iliotibial tract. Many clinical as well as experimental observations have established the fact that fascial grafts can withstand great strain. Kirschner found that whereas strips of periosteum were torn by a pull of thirteen pounds, a similar sheet of fascia lata supported a weight of ninety pounds. It exhibits an extraordinary tendency to heal in a viable state. It is readily adapted to the contours of all fields. There is no difficulty in suturing or fitting it into place.

APPRAISAL OF TRANSPLANT METHODS

Simultaneously with the evolution of the experimental phase of fascial transplantation, there developed its clinical application in a veritable host of conditions. Of interest to the orthopedic surgeon is its use in tendon defects, habitual dislocation of the shoulder, traumatic lesions of the capsule and ligaments of knee joints, flatfoot, and divided muscles such as ruptured biceps. The ophthalmic and plastic surgeons may use fascia in the treatment of paralyzed muscles as in ptosis and facial palsy. The neurosurgeon may consider its use in dural defects. The thoracic surgeon has patched pleural defects with fascia whether traumatic or postoperative, as following removal of sarcoma of the chest wall. Nephropexy by fascial strips is part of the urosurgeon's armamentarium. General surgeons are primarily interested in the use of fascial transplants in hernia and defects of the abdominal wall.

Ideal incisional hernioplasty demands an anatomic repair with free dissection and separate tier suture of all layers. This may be possible in the smaller hernias such as those following the McBurney incision. Avoidance of undue tension is the most important consideration for successful repair, since tension causes strangulation of the tissues with sloughing and failure of healing. Dickson has well expressed the importance of this factor: "It is much more important to direct effort to relieving wound tension than it is to devise some new type of suture material. A wound which is completely relaxed and without tension will have a good blood supply and no matter what suture material has been used, whether absorbable or nonabsorbable, or it might almost be said, whether any at all is used, the wound will heal."

Massive hernia with partially obliterated surrounding structures precludes anatomic repair. The number of operations that are ordinarily employed in the treatment of these cases is legion, and their very multitude is indicative of how frequently they fail; 25 to 40 per cent recur. There are many reasons for this. These cases present attenuated muscular and fascial structures with extensive scar tissue, often the re-

sult of repeated efforts to cure a postoperative hernia following infection, and such cases do not permit the ready approximation of healthy aponeurotic layers. Obesity and the generally poor physical condition of many of these patients add considerably to the risk of the operation. Under such conditions a minimal operative procedure is imperative, and yet with ordinary methods, repair of the defect would mean extensive plastic work on ill-nourished cicatricial tissues. Approximation of the edges of large defects may prove disastrous to the patient because of the sudden diminution in the size of the peritoneal cavity, which results in a disquieting increase of intra-abdominal pressure and a decrease in vital capacity. Because of this embarrassment, postoperative pulmonary complications are common. Pressure on the diaphragm produces cardiac symptoms from pressure on the heart and abdominal vessels. Acute gastric dilatation and ileus, too, are not uncommon complications. It is akin to ileus associated with a tight plaster spica and may occur whether or not the peritoneal cavity has been opened. These are the problems of hernioplasty which necessitate utilization of fascial transplants for safe and adequate repair.

The popular Gallie and Wangensteen procedures are developments of Kirschner's original and simpler patch transplantation. Both Gallie and Kirschner utilize free transplants, the former using the fascia as strips in the form of sutures. Wangensteen uses a pedicled transplant. It now behooves us to analyze the relative merits of these methods. Much light is thrown on this appraisal by a careful study of Gallie's experimental investigation as published in 1923. It was these observations which led Gallie to discard the patch transplant in favor of strips of fascia employed as interweaving sutures.

Gallie found that free transplants of fascia, whether in patches or suture strips, as well as pedicled transplants, lived unchanged when they could receive an adequate supply of lymph. This observation confirms the original work on the practicability of using free transplants of fascia. Since viability is established whether the transplant is pedicled or free, are the added technical manipulations that are part of Wangensteen's procedure at all necessary? There is no experimental evidence to prove that pedunculation of the graft ensures a greater degree of usefulness than the free graft; the value of preserving the blood and nerve supply is purely theoretical. There is no objection to using the pedicled transplant where it is readily applicable, but even Wangensteen admits that Kirschner's method of using a sheet of fascia lata as a free patch "permits of more universal application in the correction of tissue defects than the pedicled graft." For example, it may be impossible to adequately cover an upper abdominal defect with a pedicled graft, particularly in a patient with a short femur. Much of the available quantity of the graft is sacrificed by the waste entailed in covering the distance between the pedicle and the abdominal defect. This factor caused a

recurrence in one of the thirteen cases which comprised Wangensteen's original report. Another disadvantage is the possibility of obscured bleeding in the tunnel fashioned for transmission of the pedicled graft; this contrasts with the complete visualization which is always possible in using the free patch and this ensures perfect hemostasis. Finally from an aesthetic point of view, the patient occasionally objects to the cord-like bulge caused by the pedicle of the transplant and sometimes mistakes it for a recurrence of the hernia.

Gallie found that wounds in fascia, when sutured in edge-to-edge apposition with absorbable sutures, heal together by means of newly formed connective tissue which is not very strong and, if subject to strain, slowly stretches, leaving a gap between edges of the wound. He found that transplants heal to surrounding structures, by means of this delicate scar tissue, in exactly the same manner as healing of simple wounds in these tissues, and that it is the constant tendency of this scar to stretch, which explains the failure of many of the attempts which have been made to cure large hernias by the use of patch transplants. The reason for the stretching tendency of scar tissue is that, although composed of quite similar white fibers as fascia, these fibers are loosely and irregularly arranged in contrast to the parallel arrangement of the fibers in fascia which gives the latter great strength in the direction in which it is normally subject to strain. Gallie felt that he could overcome this objection by weaving the edges of the hernial defect together with the strips of fascia employed as everlasting living sutures. If approximation could not be obtained without disastrous increase in intra-abdominal pressure, Gallie originally suggested filling the gap with a living aponeurotic filigree by a basket weave of the fascial strips as in darning a sock.

At first glance one might feel that Gallie's findings doom the applicability of free patch transplants. However, we do not believe nature's process of healing wounds in aponeurotic structures to be as defective as Gallie was led to conclude. Certainly, the greatest number of laparotomy wounds heal satisfactorily with the ordinary method of suture. In those cases which do develop hernias it is everyone's experience that the latter develop soon after, or even before discharge from the hospital and not years later, as they should if stretching of the scar were the factor. Branch reported that in 46.3 per cent of 300 cases of incisional hernia, the patient noticed a mass in the wound ninety days or less after the original operation. To follow Gallie's thought to its logical conclusion, one would have to sew all laparotomy wounds with fascial suture strips. We feel that scar stretching is not the important factor in the development of postoperative herniation. More important considerations are careful closure, asepsis, and the use of incisions which cause the least trauma to muscles and nerves. Furthermore, Gallie admits that if the surfaces of the patch and surrounding tissue are thoroughly cleansed of areolar tissue, scarified and overlapped, they unite by a generous scar

and here the union is more solid and tolerates what may be considered normal degrees of strain. It is perfectly possible that it was the lack of proper cleansing and preparation of the sutured edges which accounted for the failures of some of the patch transplants, so that if these precautions are taken, uniformly good results may be obtained even without the use of Gallie's suture strips. One must bear in mind, too, that if scar stretching predisposes to failure in free transplants, this factor should apply equally in the Wangensteen pedicled transplant which is also sutured in place in the ordinary manner. And yet, the highly satisfactory results obtained with the Wangensteen procedure give cogent testimony to the successful use of transplants without fascial sutures.

Gallie found that gaping was less marked with linen than with catgut. We favor the use of silk in fixing the transplant. Since it is not absorbed, edge-to-edge apposition of transplant to environment is better maintained with a minimal tendency of stretching at the margins. Tension on the suture line is absent since the transplant method obviates the necessity of pulling the edges of the defect together, and, therefore, the sutures have no tendency to cut out. Parsons found that recurrences were four times as frequent in a series of inguinal hernias when catgut was used as when silk was employed. Whipple has shown that wounds repaired with silk were always superior in tensile strength to those repaired with catgut. Catgut caused greater trauma as well as pressure necrosis as evidenced by the zone of fluid and leucocytes surrounding the sutures.

The superiority of fascial sutures is not universally accepted. Branch reported 240 incisional hernias. Of this number, 226 repaired with fascial overlap and silk or catgut gave a recurrence rate of 19.6 per cent; 14 larger hernias were repaired by the Gallie method and of these 21.42 per cent recurred. Burdick, Gillespie, and Higinbotham reported on the use of fascial sutures in 1,485 hernias at the Hospital for Ruptured and Crippled. Their recurrence rate for 98 umbilical, epigastric, and ventral hernias was 44.89 per cent. Suppuration occurred in 7.9 per cent of the cases in which fascial sutures were autogenous. They no longer regard fascial strips as the suture material of preference and have turned to silk with better results. Gallie had to give up his filigree method of filling hernial defects because of a number of recurrences through the chinks between the sutures. He was forced to revert to the free patch transplant which he preferred to suture in place with fascial strips. His latest method is that of fixation of the patch by fashioning the latter into a many-tailed bandage design. We have not found it necessary to vary our technique at all since silk has been utilized.

A grave complication of the Gallie operation, which makes use of interweaving fascial sutures for pulling the defect margins together, is that of increased postoperative intra-abdominal pressure. Smith and Masson of the Mayo Clinic reported on the results of the use of the Gallie fascial

suture in 85 cases of ventral hernia. Four of their five postoperative deaths were due to pulmonary complications. Frequently it was necessary to place the patient in an oxygen tent immediately after operation. Power reported two cases of ileus following the Gallie operation, one of which terminated fatally.

The free patch transplant is not subject to many of the criticisms of the other plastic methods and its use deserves much more popularity. It is readily applicable to all cases. It does not have any of the limitations of the pedunculation method. It heals with a minimal reaction and is not associated with the suppuration not uncommonly present

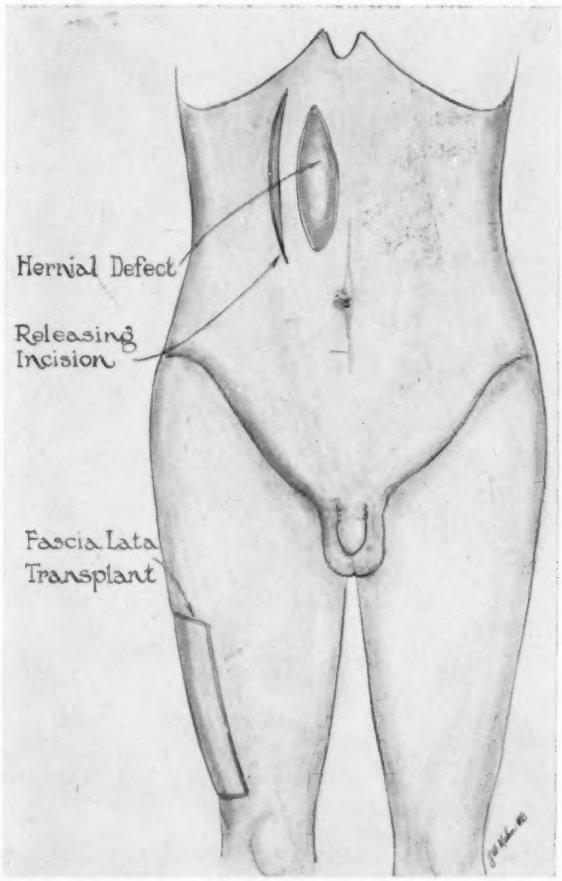

Fig. 1.—Releasing incision made through fascia on one side of hernial defect.

when fascial sutures are used. Proper preparation of the transplant and the recipient area as well as the use of silk technique ensures good healing and firm union. Wound tension and increased intra-abdominal pressure are completely avoided and this has been a primary consideration in our technique in applying the free patch.

INDICATIONS AND TECHNIQUE

The most important indication for using the fascial insert is a scarcity of satisfactory tissue about the hernial gap. Tissues that do not lend

themselves to adequate repair cannot be expected to withstand the great strain that would be put upon them, particularly with approximation under tension. Such a situation is often characteristic of recurrent hernia, large direct inguinal hernia, and massive abdominal hernia. Obviously, fascial transplantation is not indicated in every case of ventral hernia (Figs. 1, 2 and 3).

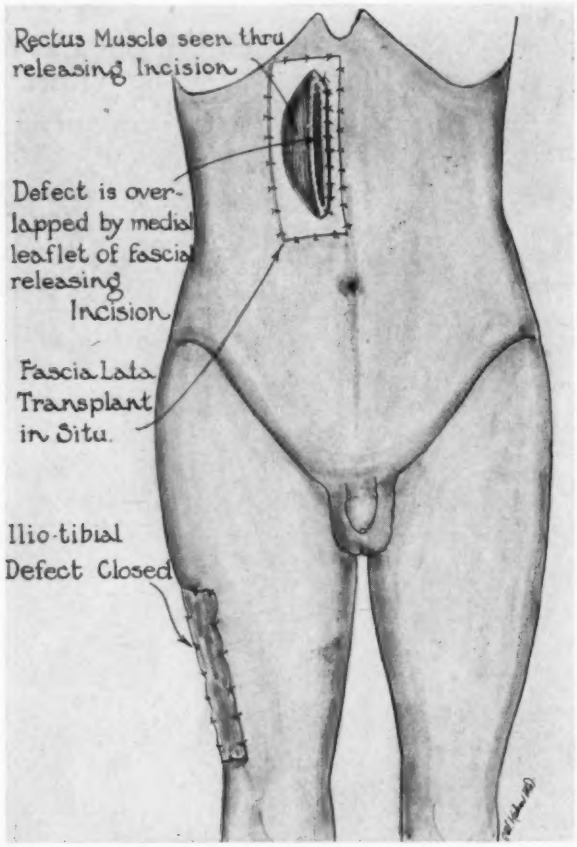
Rectus Muscle seen thru releasing Incision
Defect is overlapped by medial leaflet of fascia releasing Incision
Fascia Lata Transplant in Situ.
Ilio-tibial Defect Closed

Fig. 2.—Original defect sutured, leaving a gap at the site of releasing incision. Graft overlies entire field. Iliotibial defect is closed by suturing margins to underlying muscle.

Simplicity keynotes the technique. The peritoneal cavity need not be opened and layer dissection of the abdominal wall is unnecessary, thus also effecting a great saving of time. The defect is exposed and the surrounding fascia is cleansed of all adherent areolar and scar tissue. A releasing incision is made on one side of the defect approximately one-half inch from the margin. The peritoneal bulge is depressed in the defect and the clearly defined margins are readily sutured with interrupted black silk without any tension. The line of the releasing incision in the fascia now gapes and exposes beneath it either the attenuated muscle layer or, frequently when this is absent, merely the posterior rectus sheath or transversalis fascia. An appropriately sized sheet of

fascia lata is excised from the ilio-tibial tract of the thigh and after being properly freed of its investing areolar tissue, it is sutured over the newly made defect with interrupted black silk sutures. A large enough graft may be taken to cover the primary suture line as well, thus effecting a wide margin of overlap with actual contact between the cleansed transplant and the freshened fascial surface. We have not found it necessary to use any drains. This method permits secure closure without producing the slightest tension and thus remains true to one of the most fundamental principles of surgery. In our opinion, it is superior to all suture methods, fascial or otherwise, which pull the margins of the defect together.

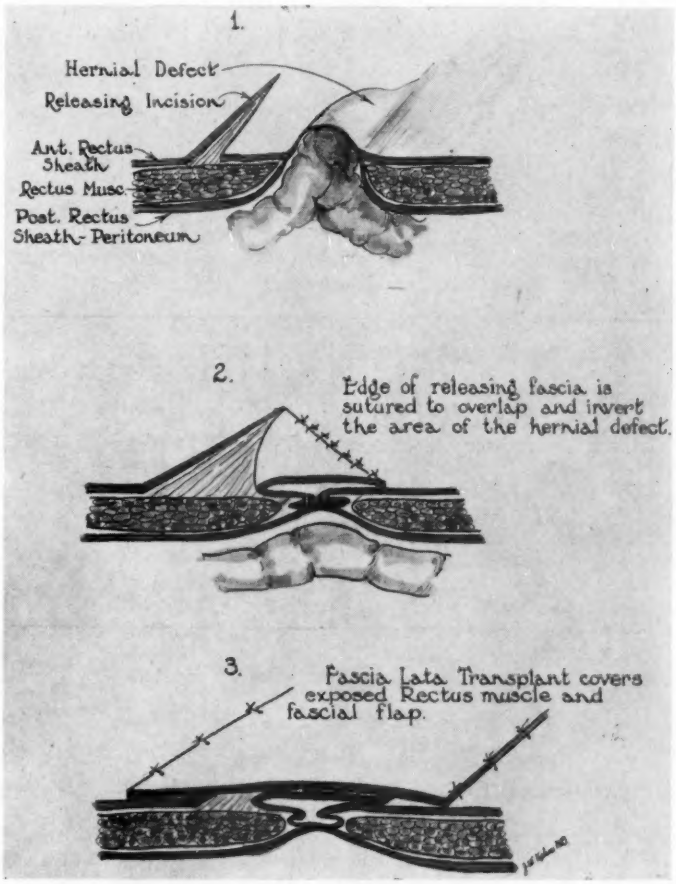
1.
Hernial Defect
Releasing Incision
Ant. Rectus Sheath
Rectus Musc.
Post. Rectus Sheath-Peritoneum
2.
Edge of releasing fascia is sutured to overlap and invert the area of the hernial defect.
3.
Fascia Lata Transplant covers exposed Rectus muscle and fascial flap.

Fig. 3.—Schematic presentation showing the steps in the repair of the hernia.

At this writing, eleven cases of large ventral hernia have been operated upon at the Jewish Hospital of Brooklyn by this method, the first one three and one-half years ago and the last about two weeks ago. This is admittedly a small group with a relatively short follow-up. However, so completely uneventful have been their recoveries and so successful the results thus far, that one cannot help but be favorably impressed.

It is the purpose of this paper to present the procedure at this time so that others may be stimulated to use it in the interest of amassing a larger group of cases for a more fully tested appraisal.

SUMMARY

The fact that one can with impunity make an autogenous transplant of fascia lata is now well established. Kirschner was the first to crystallize the method into a sound surgical procedure. The disputed question of ultimate viability of the transplant is only of academic importance. Fascia lata of the iliotibial tract is the most desirable for transplantation. Free patch transplantation of fascia deserves more popularity, since in many ways it is superior to the Gallie and Wangensteen procedures. A technique for the use of the free transplant in the surgery of large incisional hernia is described.

REFERENCES

Bassini, E.: Ueber die Behandlung des Leistenbruches, Arch. f. Klin. Chir. **11**: 429, 1890.

Branch, C. D.: Incisional Hernia; Analysis of Three Hundred Cases, New England J. Med. **211**: 949-952, 1934.

Burdick, C. G., Gillespie, C. H. M., and Higinbotham, N. L.: Fascial Suture Operations for Hernia; Summary and End-Results of 1,485 Operations, Ann. Surg. **106**: 333-343, 1937.

Dickson, A. R.: Postoperative or Ventral Hernia; Method for Relief of Tension After Repair, Surg., Gynec. & Obst. **61**: 836-837, 1935.

Gallie, W. E.: Closing Very Large Hernial Openings, Ann. Surg. **96**: 551, 1932.

Gallie, W. E., and LeMesurier, A. B.: The Use of Living Sutures in Operative Surgery, Canad. M. A. J. **11**: 504, 1921.

Gallie, W. E., and LeMesurier, A. B.: The Use of Free Transplants of Fascia as Living Sutures in the Treatment of Hernia, Tr. Am. S. A. **41**: 331, 1923.

Halsted, W. S.: The Radical Cure of Inguinal Hernia in the Male, Johns Hopkins Hosp. Bull. **4**: 17, 1893.

Kirschner, M.: Freie Sehen und freie Faszientransplantation, Verhandl. d. deutsch. Gesellsch. f. chir. **38**: 281, 1909.

Kirschner, M.: Der gegenwaertige stand und die naechsten aussichten der autoplastichen Freien Fasxienuebertragung, Beitr. z. klin. Chir. **86**: 5, 1913.

McArthur, L. L.: Autoplastic Suture in Hernia and Other Diastases, J. A. M. A. **37**: 1162, 1901.

Neuhof, H.: The Transplantation of Tissues, New York, 1923, D. Appleton-Century Company, Inc.

Parsons, W. B.: Silk Sutures in Repair of Hernia, Ann. Surg. **106**: 343-347, 1937.

Power, R. W.: Treatment of Mid-Line Ventral Hernia, Brit. M. J. **1**: 958-960, 1937.

Smith, C. H., and Masson, J. C.: Results of the Repair of Ventral Hernias With Sutures of Fascia Lata, SURGERY **7**: 204, 1940.

Wangensteen, O. H.: Repair of Recurrent and Difficult Hernias and Other Large Defects of the Abdominal Wall Employing the Iliotibial Tract of Fascia Lata as a Pedicled Flap, Surg., Gynec. & Obst. **59**: 766, 1934.

Whipple, A. O.: The Use of Silk in the Repair of Clean Wounds, Ann. Surg. **48**: 662, 1933.

CONSIDERATION OF THE FACTORS INFLUENCING THE SURGICAL REPAIR OF INGUINAL HERNIA

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ONE person in ten, the world over, is afflicted with hernia. All ages of both sexes do not escape. Ninety per cent of all hernias are inguinal. Inguinal hernia is a foremost disabling condition in industrial workers and it ranks third in the causes of rejection for military service.

Operation for the cure of inguinal hernia is a common, everyday procedure in the modern hospital. It constitutes about 10 per cent of all types of surgery done in large general hospitals. Mortality rate is less than 1 per cent and morbidity from wound infection, pulmonary and other complications is, on the average less than 10 per cent. Recurrence of hernia, as generally admitted by surgeons at the present time, is seen in 5 to 10 per cent of the cases of which 80 per cent appear within the first year following operation. That an inguinal hernia once repaired results in a recurrence is evidence of an error, particularly when a second operation effects a permanent cure. The purpose of this discussion, therefore, is to consider the many factors which influence our judgment in the surgical treatment of inguinal hernia and review certain fundamental principles that will diminish the chance of error when properly applied.

HIGHLIGHTS IN THE HISTORY OF HERNIOPLASTY

Hernia, a disease known to man since his beginning, has a story which is parallel with the history of medicine. Like so many other afflictions of mankind, rupture was an enigma through the centuries and attempts to solve its mystery by a multitude of methods were unfruitful. Credit for the first step in solving the problem of hernia is given the anatomists. Andreas Vesalius of the sixteenth century, and later Fallopius, Gimbernat, Scarpa, Cooper, and others, made discoveries in the anatomy of the inguinal region which proved to be most valuable in a fundamental understanding of inguinal hernia. The next step in unraveling the riddle of hernia was the discoveries which gave birth to the science of bacteriology and it was from Pasteur's work that Lister revolutionized surgery by the institution of his antiseptic methods in 1865. Oddly enough, the report of his first operation in 1869, using antisepsis, was a hernia case. Infection and suppuration, which had destroyed not only the surgeon's skillful work but frequently his patient as well, ceased to be the horrible result that had prevoiusly plagued the surgeon. Lister's antiseptic methods reduced surgical mortality to one-twentieth of the former rate. The enthusiasm of surgeons was manifest by the introduc-

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tion of a multitude of operations for hernia which were as numerous as the surgeons doing them. Controversy over antisepsis and asepsis soon arose but Lucas-Championnière pointed out that boiling and heat sterilization were in the true sense antiseptic though without the disadvantages of chemical antisepsis. He was the first to operate under aseptic precautions. With so many different methods attempted in the surgical treatment of hernia, there were many recurrences of hernia and it was obvious that there was a need for improvement in the technique. This came as another great step with the introduction of the Bassini[2] and Halsted[3] methods of hernia repair. The techniques were developed independently and reported at about the same time, 1890. Halsted[4] later changed his original technique so that the essential difference from the Bassini method was in relation to the spermatic cord. Bassini reconstructed the tissues posterior to the cord, transplanting it anteriorly, whereas, Halsted reinforced the tissues anterior to the spermatic cord. Strange as it may seem, these two methods have been the basis of operative repair of hernia to this day. All operations introduced have been modifications of these two original methods. New additions to the operative treatment of hernia have been made during the past fifty years and the operative technique of surgery in general has been greatly refined by knowledge gained from researches in the basic sciences as well as the surgical skill manifested by the masters of the art. Among the outstanding contributors to hernia surgery since 1890 are Wolfer,[5] who in 1892 suggested the use of the rectus muscle or its sheath as a reinforcement of Hesselbach's triangle, and Bloodgood[4-6] in the same year sutured a triangular flap of the rectus sheath to the inguinal ligament for the same reason. Andrews,[7] in 1895, described a method of imbrication of the layers of the abdominal wall in which the transversalis, internal and external oblique muscles were sutured as one layer over the cord to Poupart's ligament and the lower flap of the external oblique was carried up over the upper flap and sutured to it. This was an advancing step in the treatment of direct hernia. In 1899, Ferguson[8] closed the transversalis fascia tightly over the cord and made the internal abdominal ring smaller. McArthur,[9] in 1901, suggested the use of the edge of the external oblique aponeurosis as a living fascia suture to unite the muscle to the inguinal ligament. In 1919, LaRoque[1] devised an intraabdominal removal of the hernial sac followed by the plastic repair of the inguinal structures. Hoguet,[10] in 1920, advised opening the hernial sac near the internal inguinal ring in the repair of direct inguinal hernia, a definite advance in the surgical technique in which the direct hernial sac is converted into an indirect sac by traction on the opened peritoneum at the internal abdominal ring and dissection of the preperitoneal structures from it. Gallie and Le Mesurier,[11] in 1921, described the transplanta-

tion of fibrous tissues in the repair of anatomic defects by using fascia lata strips as suture material and graft. Pitzman,[1] in the same year, stressed the importance of repair of defects in the transversalis fascia. Seelig and Chouke,[12] in 1923, by experiments in dogs showed that muscle sutured to fascia resulted in poor union and suggested that fascial structures be sutured to fascia for the best results in the treatment of hernia. In 1934, Wangensteen[13] presented a new application of an established principle in plastic surgery, the transplantation of a musculotendinous structure, the iliotibial tract, with its nerve and blood supply intact swung up from the thigh into defects of the abdominal wall. Anson and McVay,[14] in 1938, by careful studies of the anatomy of the inguinal region, pointed out important relations of the muscle and fascia, and McVay, in 1939,[15] on the basis of these new investigations discussed a fundamental error in the Bassini operation in that the inguinal ligament does not withstand very well the pull exerted by muscles sutured to it and suggested that better results could be obtained by the suture of the inferior aponeurosis of the transversus abdominis to the fibrous covering of the pubic pecten as far laterally as the femoral vein. Anson and McVay have contributed a great deal toward the improvements that may be made in the operative repair of inguinal hernia.

REDUCTION OF THE RECURRENCE RATE

Since Halsted[4] once stated that "the cure of hernia may be listed with the triumphs of surgery," the surgical treatment has in many respects come a long way. Nevertheless, hernial surgery is far from perfect as the evidence of recurrences will bear out. Recurrent hernia in the narrowest sense implies the return of exactly the same type of hernia as existed before operation. True recurrences are relatively rare and the designation "recurrent" embraces all types of inguinal protrusions occurring after a hernioplasty. Recurrence rate among surgeons varies considerably as some quote a rate as high as 25 per cent, particularly in direct hernia, and others as low as 0.8 per cent (Joyce[16]). Undoubtedly, the personal equation plays a part. Error in technique of the original surgeon or the use of a technique not suitable for the adequate repair of the type of hernia that exists is a suggested explanation for later development of a weakness in the inguinal region that is characterized as a "recurrence." As Fallis[17] has stated, "improvement in results of hernial repair will not come from inventing new operations, but from carefully following certain well-defined principles which now yield excellent results when properly applied." To repeat that hernial surgery is not perfect, therefore, may be interpreted also as meaning that principles we have already learned in the surgical treatment are oftentimes not properly applied.

MANEUVERS IN TECHNIQUE CONTRIBUTING TO THE EASE AND THOROUGHNESS OF THE REPAIR

All hernia operations have followed certain general principles, namely, high ligation and excision of the sac, and second, plastic reconstruction of the inguinal canal. At operation, an examination of the tissues is the first step in planning the repair. Anatomic structures vary in strength and development in different individuals and obese patients frequently offer the most difficult problems. Long standing, large hernias are often accompanied by distortion and atrophy of structures essential for use in the reconstruction. Inguinal hernia in the extremes of life gives a different picture, for in the child anatomic changes to the extent of almost complete destruction have not occurred and the tissues are in a state of growth as compared to the state of degeneration in the elderly person. Depending upon the condition of the tissues, large hernias may or may not be more difficult to repair than small hernias. The accurate diagnosis of the type of hernia with which one is dealing is the next step. This can only be made by opening the peritoneum at the internal ring, introducing the examining finger through this opening into the peritoneal cavity, and testing with the finger the usual sites of exit of a peritoneal pouch such as Hesselbach's triangle and the femoral ring. Femoral hernia is in the true sense a type of inguinal hernia and is often missed entirely by omitting this important step. Failure to recognize the presence of a double sac or the saddlebag (direct-indirect) type of hernia is a principal cause of recurrence. This is likely to happen when only a small indirect hernial sac is discovered and the surgeon is less careful with repair of the small hernia than a large one. Direct hernias rarely, if ever, strangulate, but incarceration and strangulation are common in indirect and femoral hernias. The careful inspection of bowel or omentum contained within a hernial sac is the most important part of the operation and may necessitate a second incision through which the extraction, examination, and repair of the bowel is done, leaving the repair of the hernia as a step, second in importance. Most inguinal hernias contain omentum which is unattached to the sac and slips readily into the peritoneal cavity. In every operation for inguinal hernia, the loose, redundant peritoneum should be carefully withdrawn through the internal abdominal ring as the preperitoneal structures are wiped aside with the gauze-covered finger. It is surprising in many cases the amount of peritoneum that one can recover by this maneuver. That the step has a decided advantage is noted by the smoothing out of the peritoneum covering the openings from the abdominal cavity and rendering relatively loose the transversalis fascia over and about these openings making the fascia easier to repair later in the operation. As both a femoral and inguinal hernia may be present at the same time, both may be repaired by bringing the femoral sac through the opening

of the peritoneum at the internal ring as suggested by Fallis and McClure.[18] The sac should be closed at the internal abdominal ring. This may be accomplished by a continuous or purse-string suture if the opening is large or by twisting the neck of the sac and applying a transfixion suture if it is narrow. Upon excision of the sac after the closure, the stump will retract high because the peritoneum was previously drawn fairly taut. The choice of reconstruction of the inguinal region depends upon what one has found to be at fault in the supporting structures of this region. A standard operation to be used in the repair of all inguinal hernia is as impractical as a one for all procedure in any other type of surgery. Principles come to play as much importance in the repair of a hernia as in the proper correction by surgical methods of other conditions. The importance of carefully repairing weaknesses of the transversalis fascia in inguinal and femoral hernias is a step that must not be overlooked. Very often the weakness or separation of the fibers can be repaired by interrupted or plicating sutures. Where the weakness is a large one, due to wide separation of fibers, congenital absence, or atrophy, then a substitute must be made to reinforce the weakened site by the use of fascia as suggested by Gallie or the transplantation of a flap according to Wangensteen's method. The femoral opening in femoral hernia may be repaired by suturing the transversalis fascia to Cooper's ligament as suggested by Dickson.[19] To omit the repair of the transversalis fascia or to fail to apply a substitute for it is a grave error in technique and one of the chief causes of the "recurrence."

Considerable controversy has attended the wisdom of transplantation of the cord. There are those who believe that a weakness is produced at the internal abdominal ring by this step and others state that the lower inguinal region is weakened by omitting it. Here again, the choice may be made by the consideration of the facts. Gallie's statement in disagreement with the proponents of methods of repair anterior to the spermatic cord, "the time which is spent in preventing a hernia from getting out of the inguinal canal is much better spent in preventing it from getting in," is a point in favor of those methods used to repair and reinforce the structures posterior to the cord. Where there is a well-developed weakness through Hesselbach's triangle (direct hernia) and an additional support is desired over this point, the cord should be transplanted in order to conveniently suture fascial structures to fascial structures in this region. As Anson and McVay have pointed out the undesirable results of suturing muscle or fascia to Poupart's ligament, it is better to suture the inferior aponeurosis of the transversus abdominis to the pubic pecten and the insertion of the tendon of the rectus abdominis to the point of insertion of the inguinal ligament at the spine of the pubis. In addition, the corresponding portions of the aponeurosis of the internal oblique may be sutured to the pubic pecten. This accomplishes a firm reinforcement of the lower inguinal

region and transplants the cord to some extent. Where the weakness was chiefly at the internal inguinal ring, through which a large indirect sac escaped, the ring may be reinforced and narrowed by drawing together the fibers of transversalis fascia about it and the cord need not be transplanted. Where weakness was present at the internal ring, Hesselbach's triangle, and the femoral ring, steps which have been suggested may be taken to repair and strengthen all of these sites at the same operation. To me it seems that a surgeon cannot really plan his operation in the treatment of inguinal hernia any more so than he can an operation within the abdomen until he has actually made the incision and carefully examined the structures he is to deal with.

THE SELECTION OF SUTURE MATERIAL

The repair of inguinal hernia is, in most instances, a clean wound procedure as well as a problem in plastic surgery. Adherence to rules governing clean wound repair is of primary importance in hernial repair. Whipple[20] has carefully enumerated the factors of clean wound repair and healing and has placed particular emphasis on the choice of suture and ligature material.

Since Halsted[21] introduced the silk suture into American surgery in 1882, its use has become increasingly popular. His pupils became masters in the silk technique and are responsible for the place it holds in our best American clinics. Kocher,[4] in Berne, first discovered advantages of silk and Halsted later established the principles for its successful use, which have withstood the test of time as one of the great advances in surgical technique. He remarked that "the surgeon wedded to catgut is deprived of useful devices which become indispensable and a delight to the surgeon who has acquainted himself with the possibilities of silk."

The use of the silk technique stimulated the study of the effect of various suture and ligature materials in wounds and their influence upon the healing of wounds. One of the chief reasons Halsted substituted silk for catgut was that catgut was a source of infecting organisms. The present day preparation of surgical gut has eliminated the possibility of its contamination but it is known to favor growth of organisms introduced at the time of operation. Some arguments on this score may be mentioned. The length of time surgical gut remains intact and strong enough to retain anatomic structures in their position is not definitely known. Although finer strands of surgical gut with stronger tensile strength are now available, they cannot be entirely depended upon to fulfill the requirements. The strands of catgut to match silk in relative tensile strength must be larger and thus more material is left in the wound. Larger needles are required for their introduction and thereby surgical trauma to the tissues is increased. To adequately secure knots in surgical gut, the knot must be tied with greater force and the cut ends left longer to prevent the knot from

slipping. Chemicals used in the sterilization of surgical gut as well as the catgut itself tend to produce reactions in tissues. Meleney[22] showed the superiority of silk to catgut by proving less infections and more complete hemostasis, as well as ease of sterilization. He stated that the change from catgut to silk had proved to be the more important factor in the reduction of wound infection. Reid[23] has stated, "Nonabsorbable material is preferred in the ligation of blood vessels as catgut may become absorbed or stretch too rapidly, which is especially true when infection is present." Kraissl[24] has discussed suture material on a comparative basis and concluded that better general results are obtained when silk is used. Meade and Ochsner[25] in a similar study of suture materials reached similar conclusions concerning catgut and nonabsorbable sutures and showed the merits of silk and cotton thread to be desirable in repair of wounds. Howes,[26] Shambaugh,[27] Localio and Hinton,[28] Kaufman, Johnson, and Lesser,[29] and Burdick, Gillespie, and Higginbotham[30] have offered rather convincing comment on the subject of suture material to be used in hernial surgery. Ramos and Burton,[31] Zimmerman,[32] Parsons,[33] and Stein and Brown[34] have recently shown with supportive evidence the value of silk in the repair of hernia. It may be obvious, therefore, that the sutures and ligatures to be used in the repair of hernia must satisfy certain requirements. Although the ideal suture has not yet been discovered, it is wise to accept and use the suture material which will best retain the structures in their place until healing is complete if the recurrence of hernia is to be avoided.

IMPORTANCE OF THE ANESTHETIC

It is not easy to render a careful and accurate repair of an inguinal hernia if the tissues are not well relaxed. A well-chosen anesthetic to meet this requirement is essential. The agents available for this purpose are now relatively safe, particularly in the hands of trained anesthetists. The choice of anesthetic is immaterial so long as it is safe, renders complete relaxation, and is not prolonged beyond its usefulness for too long a time.

POINTS IN THE PREOPERATIVE PREPARATION AND POSTOPERATIVE CARE

In hernial surgery as in surgery in general, careful preoperative preparation of the patient as well as postoperative care contribute to the safety and comfort of the patient and the success of the operation. Except for the emergency of strangulation, hernial operation is one of election. Ample time may be taken to decrease the weight of the obese patient or to improve the nutrition of a debilitated one and thus put the tissues in their best condition. Time so taken will pay dividends. The average time required for a wound to completely heal by first intention is about thirteen days, possibly a bit less in youth and somewhat longer in the aged. It does not seem wise to allow a patient

TABLE I

SUMMARY OF THE TYPES OF HERNIA, METHODS OF REPAIR, AND THE RECURRENCES

	ANTERIOR TO CORD REPAIR; MODIFIED HALSTED TECHNIQUE	POSTERIOR TO CORD REPAIR; MODIFIED BASSINI TECHNIQUE	RECURRENCE OF HERNIA AFTER HALSTED REPAIR	RECURRENCE OF HERNIA AFTER BASSINI REPAIR
Indirect hernia	15	43	0	1 (direct)
Direct hernia	1	27	0	1 (indirect)
Bilocular hernia (direct-indirect)	0	11	0	0
Recurrent hernia	0	3	0	0
Total	16	84	0	2

Bilateral hernia was present in 26 patients. Both sides were repaired at the same time.

TABLE II

THE CASES IN WHICH RECURRENCES WERE FOUND

HENRY FORD HOSPITAL	ORIGINAL HERNIA	TECHNIQUE (YEAR)	RECURRENCE DISCOVERED	TYPE OF RECURRENCE	SECOND OPERATION
No. 208899	Bilateral indirect	Halsted, right Bassini, left Dec. 24, 1934	1941	Small direct hernia, left side	April 4, 1941
No. 219727	Bilateral direct	Bassini technique both sides July 18, 1935	1936	No true hernia; fat pad in the cord mistaken for small sac	March 17, 1936

Both recurrences followed bilateral hernioplasties.

TABLE III

DATA IN THE ONLY CASE OF WOUND INFECTION

PREOPERATIVE SKIN PREPARATION	TOTAL TIME OF OPERATION	SUTURES USED	POSTOPERATIVE HOSPITALIZATION	END RESULT
Tincture of merthiolate	40 min.	Silk	18 days	Cultured organism: Staphylococcus; small draining sinus for two months; no recurrence, 15 months

TABLE IV

POSTOPERATIVE PULMONARY COMPLICATIONS IN RELATION TO ANESTHESIA USED

	BRONCHITIS	PULMONARY ATELECTASIS	PNEUMONIA	INFARCTION	EMBOLISM	END RESULT
Ethylene and ether	0	0	0	1	0	Recovery
Avertin and ether	0	0	0	0	0	
Ether alone	0	0	0	0	0	
Spinal (Spinocaine)	0	2	0	1	0	All recovered
Total	0	2	0	2	0	

Spinal anesthesia used in 50 patients.
Inhalation anesthesia used in 24 patients (ethylene with ether 13, avertin-ether 3, ether, 8).
Local (novocain) not used in any of the cases of this series.

on his feet before his wound has been allowed to firmly heal. Some surgeons are anxious to get the patient up and about as soon as possible with the idea that he will maintain his strength and muscle tone and diminish the possibility of thrombosis and pulmonary complications. The therapeutic value of rest is not to be overlooked and a wound heals kindlier with rest than when the structures about it are put to work. Immobilization and rest of wounds not only diminishes the chance of infection but also results in a more rapid healing, as the methods of Orr[35] and Trueta[36] have shown.

The patient's occupation may be the factor to determine the length of time he is allowed to convalesce after a hernial operation. A patient whose duties require less vigorous activity, such as office work, may return to work at three weeks from the day of operation, whereas, the hard laborer should be given four or five weeks to fully regain his normal strength. This is a matter, however, which is flexible between patient and doctor, for it is doubtful if the recurrence of hernia has any bearing upon how soon the occupation is resumed if the hernia was properly repaired and the wound completely healed.

STATISTICAL STUDY OF 100 HERNIOPLASTIES

Statistical studies in a group of hernia cases are of greater value when made in a sufficiently large collection over an extended period of time. Seldom does such a study give all the facts that should be known to make it entirely accurate, for in clinical surveys many obstacles, over which one has little control, enter into the picture. The matter of keeping records is not difficult, but it is perplexing if the patient does not cooperate and return at intervals for the follow-up examination.

To eliminate to some extent the inaccuracy of a study of consecutive cases of hernia, I have not included those patients who failed to report after six months. A group consisting of 74 male laborers, in whom 100 operations for inguinal hernia was performed by me between the years 1934 to 1940, is submitted for analysis. The ages varied between 18 to 58 years. All the patients responded to recall for one year or more.

OTHER COMPLICATIONS

1. Atrophy of left testis in one patient, aged 45 years. Bassini operation was performed on both sides in repair of bilateral direct hernias.
2. Swelling of left testis without apparent atrophy in one patient, aged 53 years. Modified Halsted operation was done for repair of an indirect hernia.
3. Thrombosis of the femoral vein with tenderness and swelling of the lower extremity. No patients showed definite signs.

SUMMARY OF DATA

1. One hundred operations for inguinal hernia in 74 male laborers were performed between 1934 and 1940.

2. The cases are not consecutive, but include only those patients within this six-year period who returned for follow-up for one or more years.

3. There were 84 modified Bassini operations and 16 Halsted or anterior to the cord repairs.

4. There were two recurrences, both following Bassini repairs. A direct hernia appeared after a repair of an indirect in one case of bilateral herniotomy. The other was not a true recurrence but must be considered one technically because a large mass of fatty tissue in the cord protruded through the external ring and gave the impression of a hernial sac. The results were established at the second operation.

5. There was one case of wound infection, with a draining sinus. In this case the wound ceased to discharge after two months and remained healed for fifteen months, which was the total length of time this case was followed. When last seen there was no evidence of recurrence of hernia.

6. There were four cases of postoperative pulmonary complications from which all recovered. In one case, there was pulmonary infarction, proved by roentgenogram, following an inhalation anesthetic. Two cases of pulmonary atelectasis and one of pulmonary infarction, proved by roentgenogram, followed spinal anesthesias.

7. Spinal anesthesia was used successfully in 50 patients and an inhalation anesthetic in 24.

8. The silk technique was used in 99 operations and in one case cotton thread was substituted for silk. Since all these cases were elective, clean wound subjects, surgical gut was avoided.

9. Although there were two proved cases of postoperative pulmonary infarction, in neither case was thrombosis of the femoral vein manifested by pain or swelling in the lower extremities. The patients were discharged from the hospital on the twenty-second and twenty-ninth postoperative days respectively.

COMMENT

In this study, emphasis is placed on recurrence of hernia and factors to avoid it.

The recurrence rate of 2 per cent in this series is low and cannot be taken as a rate representing consecutive cases. More material is on hand in a study of consecutive patients but less accuracy is afforded in concluding the recurrence rate. Data taken on patients never seen after one or two months is worthless in calculating a recurrence rate. Bilateral operations are of economic value but recurrences are increased as well as complications.

Infection occurred in 1 case, or 1 per cent, in this series. The cause was suspected by discovery that silk sterilized and stored away for a few days had been used in the operation. No infections occurred be-

fore or since this event when silk or cotton thread was boiled for twenty minutes immediately before use. This point seemed to be an explanation in our case, although many avenues of contamination may be suspected as the cause of an infected wound.

Swelling and atrophy of the testis is the result of trauma to it and the spermatic cord as well as strangulation of its circulation. It may be avoided by gentle dissection and especial care in reconstructing the tissues about the internal and external inguinal rings. Overlapping of the aponeurosis of the external oblique muscle should not exert too great pressure on the cord.

Phlebothrombosis and thrombophlebitis accompanied by pulmonary infarction and embolism are complications which are infrequent. When they occur, they are potential dangers to the life of the hernia patient. One should be constantly on the alert for their early signs. It is as important to prevent them as to treat them. Operative gentleness to the tissues and complete hemostasis certainly contribute to the prevention as much as postoperative precautions. The early use of heparin in the treatment of these complications has been a lifesaving measure.

SUMMARY

The incidence of inguinal hernia and the frequency of herniotomy in modern hospitals is sited to emphasize the relative importance the disease displays in the human economy.

As a basis for the discussion of fundamental factors influencing the treatment of inguinal hernia, the highlights in the history of hernial surgery are briefly reviewed.

Although the cure of hernia is a triumph of surgery, the surgical treatment is by no means perfect as is illustrated by so-called "recurrences."

True recurrence, or the return of the exact type of hernia that existed before operation, is rare. The "recurrence" usually represents all types of inguinal protrusions that occur after hernioplasty. Reduction in the rate of recurrence may come from following well-defined principles now yielding good results when properly applied, rather than invention of new operations.

Technical steps, which contribute to the ease and thoroughness in the repair of inguinal hernia, are recalled. These steps constitute maneuvers suggested by the trail blazers in the art of hernial surgery. They are worthwhile in diminishing technical errors and this is reason enough to adopt their use.

Hernioplasty is in most cases an elective, clean wound procedure. Adherence to rules governing clean wound repair are of primary importance. The silk or cotton thread technique is within this realm and the laws for its successful use are strict, but the results obtained are the surgeon's delight.

A brief study of 100 hernioplasties, representing operations in 74 male laborers, is presented as a survey made over a six-year period. The cases were selected on the basis that all the patients responded to a recall for one year or more.

REFERENCES

1. Andrews, Edmund: History of the Development of the Technique of Herniotomy, Ann. M. Hist. **7**: 451-466, 1935; Garrison, F. H.: An Introduction to the History of Medicine, Philadelphia, 1929, W. B. Saunders Company; Thompson, Malcolm: The Operative Story of Hernia, Am. J. Surg. **43**: 162, 1939; Iason, A. H.: Hernia, Philadelphia, 1941, The Blakiston Company.
2. Bassini, Eduardo: On the Treatment of Inguinal Hernia, Arch. f. klin. Chir. **40**: 429, 1890.
3. Halsted, W. S.: Johns Hopkins Hosp. Bull. **1**: 12, 1889.
4. Halsted, W. S.: Surgical Papers, Vol. 1, Baltimore, 1924, Johns Hopkins Press, p. 261-308.
5. Turner, G. G.: Modern Operative Surgery, Ed. 2, Vol. II, Baltimore, 1935, William Wood & Company, p. 16.
6. Bloodgood, J. C.: The Transplantation of the Rectus Muscle or Its Sheath for the Cure of Inguinal Hernia When the Conjoined Tendon is Obliterated. The Transplantation of the Sartorius Muscle for the Cure of Recurrent Hernia When Pouparts Ligament Has Been Destroyed, Ann. Surg. **70**: 81, 1919.
7. Andrews, E. W.: Imbrication or Lap Joint Method: A Plastic Operation for Hernia, Chicago M. Rec. **8**: 67, 1895.
8. Ferguson, Alex. H.: Oblique Inguinal Hernia; Typical Operation for Its Radical Cure, J. A. M. A. **33**: 6, 1899.
9. McArthur, L. L.: Autoplastic Sutures in Hernia and Other Diastases, J. A. M. A. **37**: 1162, 1901; **43**: 1039, 1904.
10. Hoguet, J. P.: Direct Inguinal Hernia, Ann. Surg. **72**: 671-674, 1920.
11. Gallie, W. E., and le Mesurier, A. B.: The Use of Living Sutures in Operative Surgery, Canad. M. A. J. **11**: 504, 1921; **13**: 469, 1923; Idem. Brit. J. Surg. **12**: 289, 1924; Idem. Arch. Surg. **9**: 516, 1924.
12. Seelig, M. J., and Chouke, K. S.: A Fundamental Factor in the Recurrence of Inguinal Hernia; Arch. Surg. **7**: 553, 1923; Seelig, M. J.: Fundamental Principles Underlying the Operative Cure of Hernia, J. A. M. A. **88**: 529, 1927.
13. Wangensteen, O. H.: Repair of Recurrent and Difficult Hernias and Other Large Defects of the Abdominal Wall Employing Iliotibial Tract of Fascia as Pedicle Flap, Surg., Gynec. & Obst. **59**: 766, 1934.
14. Anson, B. J., and McVay, C. B.: Anatomy of the Inguinal and Hypogastric Regions of the Abdominal Wall, Anat. Rec. **70**: 211-225, 1938; Idem, Aponeurotic and Fascial Continuities in the Abdomen, Pelvis and Thigh **76**: 213-231, 1940.
15. McVay, C. B.: A Fundamental Error in the Bassini Operation for Direct Inguinal Hernia, Univ. Hosp. Bull., Ann Arbor **5**: 14, 1939.
16. Joyce, T. M.: Fascial Repair of Inguinal Hernias, J. A. M. A. **115**: 971, 1940.
17. Fallis, L. S.: Inguinal Hernia, Ann. Surg. **104**: 403-418, 1936; Ibid **106**: 363, 1937; Idem, Postoperative Wound Disruption, SURGERY **1**: 523, 1937; Idem, Direct Inguinal Hernia, Ann. Surg. **107**: 572, 1938.
18. McClure, R. D., and Fallis, L. S.: Report of 90 Operations for Femoral Hernia, Ann. Surg. **109**: 987, 1939.
19. Dickson, A. R.: Femoral Hernia, Surg., Gynec. & Obst. **63**: 665-669, 1936.
20. Whipple, A. O.: The Choice and Use of Ligature and Suture Material in the Repair of Clean Wounds, Surg., Gynec. & Obst. **69**: 109, 1939; Idem, Use of Silk in Repair of Clean Wounds, Ann. Surg. **98**: 662, 1933; Idem, The Essential Principles in Clean Wound Healing, Surg., Gynec. & Obst. **70**: 257, 1940; Idem, The Critical Latent or Lag Period in the Healing of Wounds, Ann. Surg. **112**: 481, 1940.
21. Halsted, W. S.: The Employment of Fine Silk in Preference to Catgut and the Advantages of Transfixing Tissue and Vessels in Controlling Hemorrhage, J. A. M. A. **60**: 1119, 1913.
22. Meleney, F. L.: Infection in Clean Operative Wounds, Surg., Gynec. & Obst. **60**: 264, 1935.

23. Reid, M. R.: The Ligation of Large Arteries, Surg., Gynec. & Obst. **58**: 287-296, 1934.
24. Kraissl, C. J.: Suture Material: A Review of Recent Literature, Internat. Abstr. Surg. **62**: 417, 1936; Kraissl C. J., Keston, B. M., and Cimiotte, J. G.: Relation of Catgut Sensitivity to Wound Healing, Surg., Gynec. & Obst. **66**: 628, 1938.
25. Meade, W. H., and Ochsner, Alton: The Relative Value of Catgut, Silk, Linen and Cotton as Suture Materials, SURGERY **7**: 485, 1940.
26. Howes, E. L.: The Immediate Strength of the Sutured Wound, SURGERY **7**: 24, 1940; Idem, How to Use Catgut, Surg., Gynec. & Obst. **73**: 319, 1941.
27. Shambaugh, P.: Postoperative Wound Complications. A Clinical Study With Special Reference to the Use of Silk, Surg., Gynec. & Obst. **64**: 765, 1937; Idem, The Silk Technique, SURGERY **7**: 9, 1940.
28. Localio, S. A., and Hinton, J. W.: The Choice and Use of Cotton for Suture Material, Surg., Gynec. & Obst. **72**: 615, 1941.
29. Kaufman, L. R., Johnson, W. W., and Lesser, A.: Clinical Study of Alloy Steel Wire Sutures in Hernia Repair, Surg., Gynec. & Obst. **69**: 684, 1939.
30. Burdick, C. G., Gillespie, D. H. M., and Higginbotham, N. L.: Fascial Suture Operations for Hernia, Ann. Surg. **106**: 333, 1937.
31. Ramos, R. L., and Burton, C. C.: Inguinal Hernia: Application of Cardinal Principles in the Repair of Inguinal Hernia, Surg., Gynec. & Obst. **69**: 688, 1939; Idem, The Results of the Treatment of Bilocular and Direct Inguinal Hernias, Surg., Gynec. & Obst. **70**: 953, 1940.
32. Zimmerman, L. M.: Essential Problems in the Surgical Treatment of Inguinal Hernia, Surg., Gynec. & Obst. **71**: 654, 1940.
33. Parsons, W. B.: Silk Sutures in the Repair of Hernia, Ann. Surg. **106**: 343, 1937.
34. Stein, J. J., and Brown, P. F.: The Repair of Inguinal Hernia. A Standardized Technique, Surg., Gynec. & Obst. **70**: 1054, 1940.
35. Orr, H. W.: Treatment of Osteomyelitis and Other Infected Wounds by Drainage and Rest, Surg., Gynec. & Obst. **45**: 446, 1927.
Orr, H. W.: Osteomyelitis in Compound Fractures and Other Infected Wounds; Treatment by the Method of Drainage and Rest, St. Louis, 1929, C. V. Mosby Company.
36. Trueta, J.: Treatment of War Wounds and Fractures, New York, 1940, Paul B. Hoeber, Inc.

INTESTINAL CAPILLARY CIRCULATION DURING DISTENTION

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RECOVERY of normal activity in obstructed and distended intestine is possible after many hours.[1, 2] The mortality rate of distention is associated intimately with the degree of embarrassment of the blood supply to the affected portion of the bowel.[3] The question of viability of the intestine[3] is important and the present study was undertaken to determine the role of the intestinal capillary circulation during varying degrees of intraluminal pressure.

METHOD

Rats anesthetized with pentobarbital sodium subcutaneously (4.5 mg. per 100 Gm. rat) were used. A fourth of this dose was repeated hourly as needed. A 2 cm. piece of intestine (jejunum or ileum), drawn out through an abdominal stab wound, was distended with air and illuminated without heating[4, 5] (Fig. 1), but with blood supply intact. Capillary circulation was observed at a magnification up to 150 diameters.

The field under observation and all exposed viscera were kept covered with a gentle stream of mammalian Ringer's solution at 38° C. The rat itself was supported on a warm stage, also at 38° C. It proved important to prevent angulation or kinking of the vessels supplying the distended loop. This factor was controlled uniformly.

Careful hemostasis was observed throughout. The ligatures holding the cannula for air pressure and the quartz rod light source (Fig. 1) were placed so that the blood supply to the loop of intestine under consideration was not interrupted. A loop was chosen so that it was supplied by both limbs of a mesenteric arterial arcade of vessels.

Controls with loops prepared as described but without distention did not show any change of capillary circulation up to five hours. This controls the factor of necessary exposure.

Illumination within the intestine was furnished by a modification of the Knisely technique[4] as described by Wakim and Mann.[5]

Two series of experiments were performed: (1) increasing degrees of distention for short periods (two minutes) to determine the level at which intraparietal capillary flow was changed or stopped; (2) increasing time periods during which the intestine was maintained at a fixed distending pressure. Capillary circulation was observed during and at the end of this second series of experiments up to thirty to sixty minutes after deflation.

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RESULTS

Description of the Capillary Field.—In any field examined at the antimesenteric border under a magnification of 75 to 150 diameters (Fig. 2) several arterioles and venules were seen accompanying one another, each pair lying only approximately at right angles to the long axis of the gut. The capillaries may be seen issuing from the arterioles. They always arise from their vessel of origin so as to lie either strictly in the long axis or at 90 degrees to the long axis. Thus the typical capillary pattern, as seen, described a series of rectangles (the sides of which were often at different levels) which overlay or underlay the larger vessels. This pattern is especially evident when for any reason the capillaries are dilated (reactive hyperemia) or when they contain motionless erythrocytes (excessive intraluminal distending pressure).

Continuous examination of any field made it quite evident that the smallest vessels were of two kinds, one which always was carrying blood and another which showed only an intermittent flow. Both types showed plasma skimming.

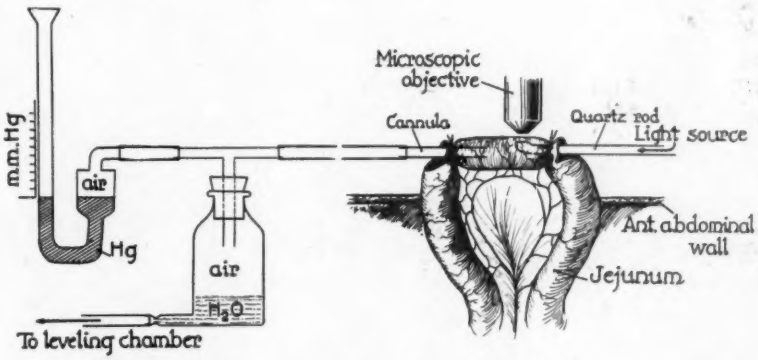

Fig. 1.—Experimental assembly for visualizing intraparietal intestinal circulation during intraluminal distention.

When the circulation through capillaries was viewed within a lymph aggregate, the usual pattern of distribution was not seen. No definite plan of distribution could be distinguished within the node.

Intraluminal Pressures at Which Intestinal Intraparietal Capillary Circulation Is Altered.—At distending pressures of 10 and 20 mm. of mercury the intestine was more transparent than before distention but no changes of circulation were noted. At 30 mm. the capillary circulation was at first a little slower, then faster again, as was the circulation in the arterioles and venules. Individual erythrocytes could be identified. At 40 mm. a few capillaries disappeared and a few more contained motionless erythrocytes, but most were normal. Flow in arterioles and venules was slower. At 50 mm. only a few capillaries were patent. Some larger vessels contained oscillating cells while the largest still had rapidly flowing blood. At 60 mm. all capillary flow had ceased and only in the largest vessels was blood flowing rapidly.

At 70 mm. no flow at all was seen except in one or two large vessels (arterioles and venules) which had a sluggish or oscillating flow and at 80 mm. even this had stopped.

At each distending pressure up to 50 mm. of mercury the rate of flow immediately after establishment of the new level was slower than previously but within one minute it was faster again. At the end of any series when the distending pressure was released, intense hyperemia was seen, the so-called reactive hyperemia. Functioning capillaries were dilated widely. The typical capillary pattern was thus quite clear and many capillaries could be seen that had not been in evidence before. This hyperemia lasted less than one minute.

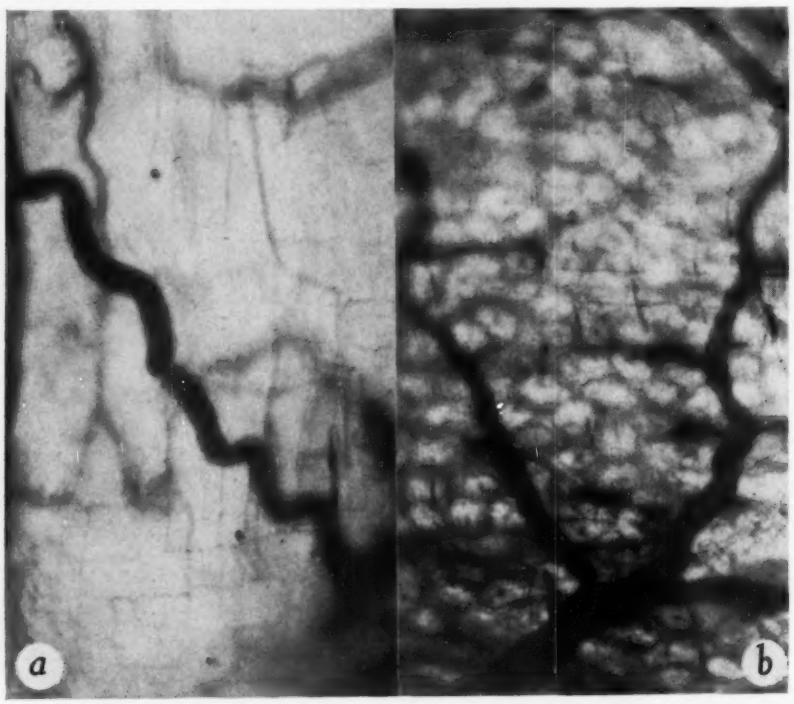

Fig. 2.—(*a*) Typical capillary pattern in undistended intestine; (*b*) same during an intraluminal distending pressure of 30 mm. of mercury (×100).

Although capillary flow no longer could be seen at levels of 60 mm. or more distending intraluminal pressure, there was active circulation in the parent arteriole and venule with pressure as great as 90 mm. These vessels which continued active could be traced backward into the mesentery.

Varying Times of Distention at a Constant Intraluminal Distending Pressure.—At a distending pressure of 15 mm. the intestinal wall was rendered somewhat more transparent than before distention. Capillary circulation remained unchanged for periods up to five hours. Deflation was followed by reactive hyperemia of less than one-minute duration.

At a distending pressure of 30 mm. capillary flow in most cases was somewhat slower than before distention (individual corpuscles could be distinguished) but remained good (Fig. 3). This adequate capillary flow persisted for periods tested up to five hours. Flow in the larger vessels was proportionately slower. The intestinal wall was transparent. In one case in which distention was carried to five hours, flow came to an end in the capillaries at 3.5 hours but remained good in larger vessels. Deflation in all cases (including the one in which capillary flow stopped) was followed by intense dilation of all vessels and appearance of some new capillaries. In the case in which capillary flow stopped, some capillaries with stationary erythrocytes were seen thirty minutes after release of pressure. Scattered ecchymoses were also present in this case.

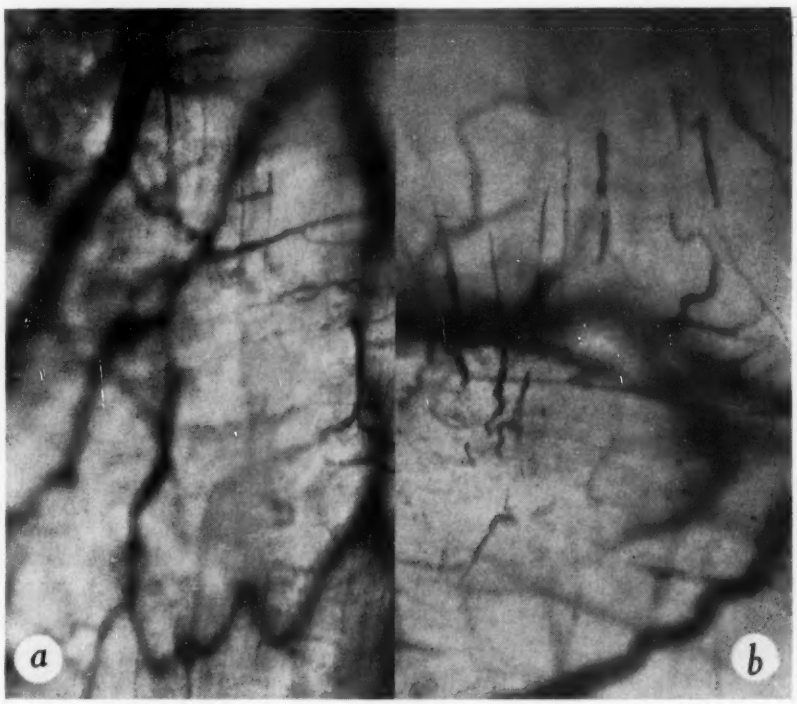

Fig. 3*a* and *b*.—Reactive hyperemia during deflation after intraluminal distending pressure of 30 mm. of mercury (×100).

At higher distending pressures of 50 to 60 mm. of mercury, studied for periods of one to two hours, capillary flow was diminished in rate. This slowing of rate was soon followed by cessation of flow, which condition persisted for one to two hours. After deflation capillary flow recovered in large part. However, a few capillaries were plugged and some ecchymoses were present at the end of one hour.

Peristalsis.—Peristalsis was uniformly absent in the loop of intestine under study during the period of distention. It returned with vigor

following deflation. Adjacent loops, above and below the distended one, were frequently active during the period of distention in the test loop.

Role of Kinking of Extraparietal Vessels.—It was found that the loop of intestine studied must be long enough to prevent angulation or kinking of vessels of supply during the time of inflation. When this angulation was prevented at a distending intraluminal pressure of 30 mm. of mercury the intraparietal capillary circulation always remained good. When kinking or angulation was permitted, flow sometimes ceased in capillaries with clumping of erythrocytes and ecchymoses, even at a distending intraluminal pressure of 30 mm. Despite this, recovery of flow took place in most of these capillaries following deflation. Nevertheless, some capillaries remained plugged thirty minutes later.

Histologic Controls.—Sections taken from the distended test segment of intestine and control segments situated approximately 2 cm. proximal were stained and compared. At distending pressures of 15 and 30 mm. of mercury maintained for five hours there were no distinguishing histologic changes.[2] At higher distending pressures (50 to 60 mm.) maintained for two hours changes were seen, consisting principally of hemorrhages beneath the visceral peritoneum and into the muscular layers and mucosa. Large dilated vessels plugged with erythrocytes and disintegration of the mucosa were also noted as a result of these high distending pressures.

COMMENT

Landis[6] has pointed out that pressures in the capillaries of rat mesentery have an average value of 12.6 mm. of mercury at the venous end. At first glance one might think that an opposing distending intraluminal pressure about 13 mm. would bring capillary flow to a standstill. However, in these experiments intraluminal pressures of four to five times that value were required to bring capillary flow to an end. This may be due either to the fact that the gradient of pressure from capillary to vein[7] persists during intraluminal distention since such pressure probably is applied equally to intraparietal arterioles, capillaries and veins; or it may be that the intraluminal distending pressure is not entirely brought to bear on the vessels of the wall. It is possible that both these factors may operate.

A continuous flow in arterioles and venules at distending pressures of 50 to 60 mm. of mercury, although blood is no longer circulating in capillaries, may indicate the presence of arteriolovenular anastomoses, which have been described in the mesentery of the mouse[8] just proximal to the capillary bed or continuity of the two vessels. The improvement of flow after an initial slowing at any new distending level is similar to that which has been shown in dogs by different methods.[9] Deflation hyperemia also has been demonstrated by different technics.[10]

Maximal intra-enteric pressure with simple experimental obstruction and in clinical cases without peristalsis is approximately 15 mm. of mercury.[11] Since at this value our experiments did not indicate any interference with capillary circulation for periods up to five hours, a basis is furnished for understanding the success with which obstruction of the intestine with distention is endured by the organism. Maximal intra-enteric pressure found in clinical cases with peristalsis is 23 mm.[11] Our series at 30 mm., representative of this group, indicated that capillary circulation usually is not obstructed by distention for as long as five hours and even though it may be, recovery of most capillaries after deflation is the rule.

In closed obstruction, higher values (50 to 60 mm. of mercury) for intraluminal distending pressures occur.[12] At these pressures in our experiments capillary flow ceased. However, it recovered after two hours of distention although ecchymoses and a few plugged capillaries were to be found after thirty minutes of observation following deflation.

Angulation of extraparietal vessels, ruled out in this study, must be an important additional factor in clinical cases. Observations during this study confirm this fact. Reactive hyperemia following deflation may be similar to the favorable sign surgeons seek at the operating table following deflation, indicating viability.

It is of interest that the capillaries lie at right angles to one another, to the transverse, and to the long axis of the gut. It may be suggested that their relation to circular and longitudinal muscle fibers is concerned in this pattern. The presence of two kinds of capillaries has been described already.[8]

SUMMARY

A typical pattern for intestinal intraparietal capillaries is described. Intraparietal capillary flow ceases when intraluminal distending pressure reaches 50 to 60 mm. of mercury. At an intraluminal distending pressure of 15 mm. maintained for five hours no change of intraparietal capillary circulation was detected. At an intraluminal distending pressure of 30 mm. maintained for five hours intraparietal capillary circulation was slightly slower than normal but remained adequate. At an intraluminal distending pressure of 50 to 60 mm. maintained for two hours intraparietal capillary flow ceased within one hour. Reactive hyperemia followed deflation in all series of distentions studied. If angulation or twisting of extramural vessels is permitted, intraparietal capillary flow stops at low values of distending pressure (less than 30 mm.).

REFERENCES

1. Aird, Ian: Effect on Blood Pressure of Sudden Release of Intestinal Distension, Proc. Soc. Exper. Biol. & Med. 32: 1593-1595, 1935.
2. Gage, Mims, and Hosoi, Kiyoshi: Histological Changes Observed in Intestinal Wall Following Simple Mechanical Obstruction in Rabbits, Proc. Soc. Exper. Biol. & Med. 32: 1651-1653, 1935.

3. Mayo, C. W., Miller, J. M., and Stalker, L. K.: Acute Intestinal Obstruction, Surg., Gynec. & Obst. **71**: 589-598, 1940.
4. Knisely, M. H.: A Method of Illuminating Living Structures for Microscopic Study, Anat. Rec. **64**: 499-524, 1936.
5. Wakim, K. G., and Mann, F. C.: Intrahepatic Circulation of Blood, Anat. Rec. **82**: 233-253, 1942.
6. Landis, E. M.: Capillary Pressure and Capillary Permeability, Physiol. Rev. **14**: 404-481, 1934.
7. Landis, E. M.: The Capillary Pressure in Frog Mesentery as Determined by Micro-injection Methods, Am. J. Physiol. **75**: 548-570, 1926.
8. Zweifach, B. W., and Kossmann, C. E.: Micromanipulation of Small Blood Vessels in the Mouse, Am. J. Physiol. **120**: 23-35, 1937.
9. Lawson, Hampden, and Chumley, Jack: The Effect of Distension on Blood Flow Through the Intestine, Am. J. Physiol. **131**: 368-377, 1940.
10. Lawson, H.: The Mechanism of Deflation Hyperemia in the Intestine, Am. J. Physiol. **134**: 147-156, 1941.
11. Sperling, Louis, Paine, J. R., and Wangensteen, O. H.: Intra-enteric Pressure in Experimental and Clinical Obstruction, Proc. Soc. Exper. Biol. & Med. **32**: 1504-1506, 1935.
12. Owings, J. C., McIntosh, C. A., Stone, H. B., and Weinberg, J. H.: Intra-intestinal Pressure in Obstruction, Arch. Surg. **17**: 507-520, 1928.

CHRONIC PARTIAL INTESTINAL OBSTRUCTION DUE TO INTUSSUSCEPTION OF AN APPENDIX EPIPLOICA

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THE unusual pathologic picture presented at operation and the interpretation of the course of events in the clinical history prompted the report of the following case.

CASE REPORT

P. H. (No. 486158), aged 56 years, a white, married man, noted a marked change in his usually very regular bowel habits during the twelve weeks prior to admission. This was accompanied by mild left lower quadrant abdominal discomfort; however, the constipation during this period was severe and required the constant use of cathartics. The stool was small, dry, and thin. Slight bloodstreaking of the stool was noted on a few occasions. There was free passage of flatus but no nausea or vomiting. Weight loss amounted to four pounds.

Examination revealed a well-nourished adult with two positive findings: a loop of distended bowel in the left lower quadrant, with visible peristalsis. There was no mass, tenderness, or rigidity.

Sigmoidoscopy.—Intestinal mucosa was normal as far as the end of the instrument.

Roentgenography.—Study by means of barium enemas, repeated on three occasions, revealed complete obstruction at the mid-sigmoid, with a large filling defect at that point. These findings, in the opinion of the roentgenologist, were typical of carcinoma (Fig. 1).

Diagnosis.—Chronic partial intestinal obstruction due to carcinoma was the preoperative diagnosis. Polypoid tumors (polyp, submucous lipoma, etc.), or specific granulomas (actinomycosis, gumma, tuberculoma, lymphogranuloma inguinale)[1] might cause the picture presented by this patient but such diagnoses were not seriously entertained.

Operation.—The abdomen was entered through a left lower rectus muscle-splitting incision. Exploration revealed a puckering of the posteromedial wall of the sigmoid with what, at first, was thought to be a submucous lipoma about 3 cm. in diameter, almost completely obstructing the lumen of the bowel. The proximal colon was dilated; the distal colon was normal. No lymph nodes were palpable in the mesosigmoid. The liver was normal; several stones were present in the gall bladder.

Closer inspection proved the lesion to be an invagination, or, better, an intussusception of an epiploic appendage into the bowel causing an almost complete obstruction (Fig. 2). Palpation through the wall of the bowel indicated to the surgeon that the mucosa was ulcerated (Fig. 3). There was no proof that the mucosal lesion was benign; it was decided, therefore, to excise the segment of bowel bearing the lesion. Accordingly, an obstructive resection was performed.

The postoperative course was uneventful. The spur was crushed on the thirteenth postoperative day; the colostomy was closed on the thirty-second postoperative day, and the patient was discharged on the sixtieth postoperative day.

Pathology.—Gross: Specimen consisted of a resected portion of the sigmoid which measured 10 cm. in length. The serosal surface was smooth. In the center of the specimen in the region between the anterior and lateral taenia coli there

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was a puckered mass which had carried along with it an epiploic appendage. On section the part which had become invaginated consisted of a partially necrotic mass which was roughly spherical in shape and measured 3.5 cm. in diameter. The mucosal surface was greenish and necrotic at the head of the mass but at the base

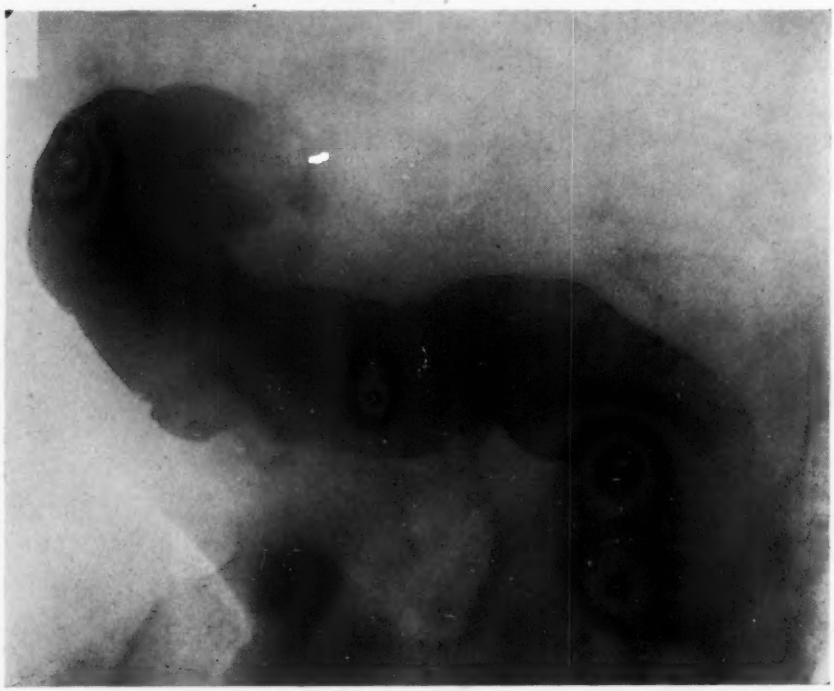

Fig. 1.—X-ray of barium enema, showing filling defect and obstruction at the mid-sigmoid. (Oblique view.)

Fig. 2.—Appendix epiploica jutting out of the "burrow" created for it by the bowel wall.

the mucous membrane was granular and moderately inflamed. This mass had apparently become intussuscepted into the colon. The remainder of the mucous membrane showed no gross abnormalities. The distance between the tumor mass and

one line of resection was 3 cm., between the tumor mass and the opposite line of resection, 4.5 cm.

Microscopic.—On section the mass consisted of fat tissue which was connected with epiploic appendage and its surface was covered by thinned out muscle layer of intestinal wall.

Analysis of Pathology.—As can be seen (Fig. 2) the epiploic appendage had become intussuscepted into the sigmoid, completely surrounded by the bowel wall. This wall of the invaginated sac was composed of the normal number of layers, but the muscularis was markedly thinned (Fig. 4). The overlying mucosal surface had a chronically ulcerated base as well as acute ulceration (Fig. 3).

Fig. 3.—Bowel opened to expose mucosa. Mucosal ulceration may be seen at lower center of photograph.

Fig. 4.—Hemisection of bowel wall and epiploic appendage (gross). The bowel wall is intact although the muscle layer is markedly thinned out over the apex of the invaginated appendage.

No similar case could be found reported in the literature. The surgical importance of the appendices epiploicae has been the subject of relatively few dissertations. Littré, in 1703, is believed to have recorded

the first case with a discussion of these fatty appendages, when he found a pea-sized, white, stonelike foreign body in the abdomen of a cadaver, a free appendix epiploica.[2] In 1917, Harrigan[3] reviewed the literature to that time; since then, several reviews have appeared namely, Hunt (1919),[4] Klingenstein (1924),[5] Patterson (1933),[6] and Moore (1940).[7] Patterson[6] divided the diseases affecting the appendices epiploicae into four categories: inflammatory changes, torsion of the pedicle, calcification and formation of loose bodies, and incarceration in a hernial sac with or without torsion. Moore[7] subclassified certain of these groups, but, in all, the only mention made of intestinal obstruction, which in any way concerned the appendages, has been in connection with inflammatory adhesive bands either between appendages or from one to another organ or the parietes. A total of ten such cases[8] has been collected. However, no report of invagination of an appendage into the bowel could be discovered in the literature.

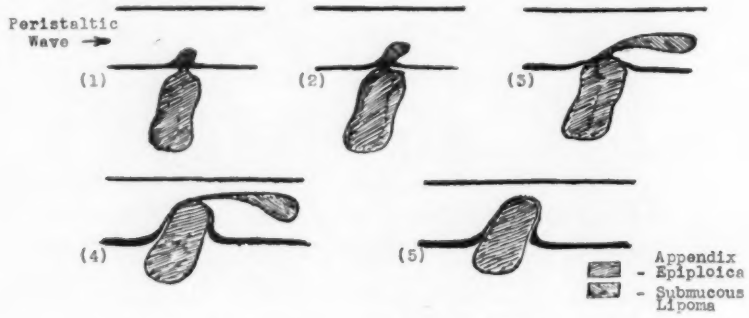

Fig. 5.—Schema of suggested pathogenesis of lesion.

It is of interest to speculate upon the etiology of this unusual picture. A congenital weakness may always be offered as a possibility, although there is no proof of it; no similar example has been previously reported. To account for the invagination one must postulate a series of unknown pressure factors within the abdomen since the intralumenal pressure due to repeated passage of firm, semisolid feces forced along by peristalsis would more likely cause an outpouching. Such an outpouching may more properly be called a diverticulum, a true diverticulum, since all the bowel coats would be involved. It may be recalled that congenital weakness is mentioned as one of the possible etiologic factors underlying the formation of diverticula (Beer,[9] Telling,[10] McGrath,[11] quoted by Wilkinson[12]).

A second and more generally accepted theory than the one mentioned above for the origin of colonic diverticula postulates that they tend to form at the base of an epiploic appendage, pushing out along a potential weakness created by the exitus of the blood vessels through the muscle coat of the bowel (Graser,[13] Wilkinson,[12] Patterson,[14] Grado[15]; and Bland-Sutton[16] quoted by Telling[10] and Hunt[4]). The sigmoid is probably the most common of all parts of the colon for diverticula to occur

(Wilkinson,[12] Spriggs and Marxer,[17] and MacCallum[18]). Their size may vary from that of a pea to the dimension of a hen's egg (McGrath[11]). However, its invagination cannot be easily explained. On the contrary, there usually occurs a narrowing of the neck of these outpouchings which would tend to prevent the invagination that occurred in the case reported. The accepted theory concerning the formation of colonic diverticula and the generally known complications associated with their presence are sufficient reasons for considering an invaginated diverticulum in our case as an untenable explanation.

A plausible pathogenesis has been offered by Dr. S. Otani, Associate Pathologist to the Mt. Sinai Hospital. He suggests that a submucous lipoma (or polyp) may originally have been present, attached to the bowel wall at the site of the ulcerated mucosa covering the invaginated portion. The dragging action of the fecal stream upon this growth gradually increased its length and narrowed the neck of the pedicle, at the same time tending to invaginate the bowel wall at its base. This could weaken the muscle layer of the wall at the area subjacent to the base of an epiploic appendage and, thus, invaginate the appendage. Continued pull of the fecal stream could pull off the polypoid growth, leaving an ulcerated mucosal base which persisted (Fig. 5).

Manheim and Peskin[19] recently reported a case in which a submucous lipoma of the sigmoid was extruded spontaneously; in addition, they collected twenty similar cases from the literature. They state, "One theory offered is that invagination of the affected area of the intestine results in necrosis with sequestration of the intussuscepted portion." This theory would confirm Dr. Otani's theory if it could be assumed that the intussuscepted portion was not sequestrated. However, we have no proof that this polypoid growth ever was present, but mention this interesting and hypothetic possibility.

Summary.—There is presented herewith the case history of a patient suffering from chronic partial intestinal obstruction caused by the intussusception of an epiploic appendix into the sigmoid, which, clinically, was indistinguishable from an obstructing neoplasm. The possible causes of this unusual condition are discussed.

ADDENDUM

Since this report was accepted for publication, three additional cases of intestinal obstruction caused by epiploic appendages were reported by Giffin, H. M., Mc Manamy, E. P., and Waugh, J. M.: Arch. Surg. **45**: 351, 1942.

REFERENCES

1. David, Vernon C.: Surg., Gynec. & Obst. **64**: 281, 1937.
2. Littré: Quoted by Harrigan,[3] L'Histoire de L'Academie Royale des Sciences, 1703, pp. 36.
3. Harrigan, A. H.: Ann. Surg. **66**: 467, 1917.
4. Hunt, V. C.: Ann. Surg. **69**: 31, 1919.
5. Klingenstein, Percy: Surg. Gynec. & Obst. **38**: 376, 1924.
6. Patterson, Daniel C.: New England J. Med. **209**: 1255, 1933.

7. Moore, George A.: New England J. Med. **222**: 919, 1940.
8. Riedel: München. med. Wchnschr. **52**: 2308, 1905 (2 cases).
Klingenstein:[5] 1 case.
Patterson:[6] 1 case and 2 cases collected from literature.
McIver, M. A.: Am. J. Surg. **19**: 163, 1933 (1 case).
Baumeister, C., Hargens, C. W., and Morsman, C. F.: Ann. Surg. **107**: 153, 1938 (1 case).
Moore:[7] 2 cases.
9. Beer, Edwin: Am. J. M. Sc. **128**: 139, 1904.
10. Telling, W. H. M.: Lancet **1**: 843, 928, 1908.
11. McGrath, B. F.: Surg., Gynec. & Obst. **15**: 429, 1912.
12. Wilkinson, S. A.: New England J. Med. **209**: 197, 1933.
13. Graser: Arch. f. klin. Chir. **59**: 638, 1899.
14. Patterson, D. C.: Tr. New England. S. Soc. **20**: 201, 1937.
15. Grado: Arch. di radiol. **7**: 1140, 1931.
16. Bland-Sutton, J.: Lancet **2**: 1148, 1903.
17. Spriggs, E. I., and Marxer, A. O.: Quart. J. Med. **19**: 1-35, 1925.
18. MacCallum, W.: Textbook of Pathology, Philadelphia, 1936, W. B. Saunders Company, pp. 412.
19. Manheim, Sylvan, and Peskin, H.: J. A. M. A. **118**: 1214, 1942.

THE MICROSCOPIC DIAGNOSIS OF RADIOPAQUE SUBSTANCE IN THE VERMIFORM APPENDIX

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IN 1937, the curiosity of one of us was aroused by a peculiar content, noted through the microscope, in the lumen of an appendix which had been sectioned following removal at operation. The content was interpreted as radiopaque substance, received by the patient at some time for roentgen examination. Subsequent clinical, radiographic, and pathologic study confirmed such interpretation. The content was apparently barium sulfate. Gross[1] and spectrophotometric[2] detection of barium sulfate in the extirpated appendix has been recorded in the literature. Reference, however, to observation and study identical with ours does not appear in the *Quarterly Cumulative Index Medicus* of the past twenty-five years. For this reason, as well as for the implications which it carries for pathologist, roentgenologist, and surgeon, the microscopic diagnosis of radiopaque substances, particularly barium sulfate, in the appendix is here described.

REPORT OF CASES

From Oct. 1, 1940, to Sept. 30, 1941, 854 appendices were examined histologically at the Buffalo General Hospital. In the following ten specimens, what appeared to be barium sulfate was found microscopically.

Case 1.—F. M., was a girl, 21 years old. The preoperative diagnosis was chronic appendicitis. Nov. 7, 1940, gastrointestinal series and barium enema were given. The appendix was visualized and Nov. 14, 1940, appendectomy was done.

Case 2.—M. N. was a 15-year-old girl. The preoperative diagnosis was ruptured corpus luteum or chronic appendicitis. Dec. 2, 1940, gastrointestinal series was made. The appendix was visualized in a retrocecal position; it was fixed by external adhesions, tender. Dec. 4, 1940, appendectomy was done; appendix retrocecal.

Case 3.—J. L., was a male, 21 years old. The preoperative diagnosis was appendicular colic. Nov. 29, 1940, gastrointestinal series was made. The appendix was faintly visualized, and found incompletely filled as a result of fecaliths, tender. On Dec. 2, 1940, appendectomy was done. The appendix was dilated by fecaliths.

Case 4.—L. C. was a 43-year-old woman. The preoperative diagnosis was gastric ulcer, uterine fibroids, and chronic appendicitis. On Dec. 9, 1940, roentgen examination of the stomach and duodenum was made and gastric ulcer and duodenal diverticulum were found. On Dec. 10, 1940, resection of ulcer, hysterectomy, left salpingo-oophorectomy, and appendectomy were done. The appendix contained fecalith; chronic appendicitis.

An abstract of this paper was read before the Buffalo Pathological Society, May 11, 1942.
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CASE 5.—L. D., was a woman, 40 years old. The preoperative diagnosis was fibroid uterus. On Feb. 25 to 28, 1941, gastrointestinal series and barium enema were given. Esophageal diverticulum was found. The appendix was visualized. On March 8, 1941, hysterectomy, left salpingo-oophorectomy, and appendectomy were done. The appendix was adherent, lumen obliterated in the distal part, and chronic periappendicitis.

CASE 6.—R. S., was a 57-year-old man. The preoperative diagnosis was peptic ulcer with perforation. March 4, 1941, gastrointestinal series was started but stopped because the patient developed pain. March 11, 1941, his appendix was obtained at autopsy.

CASE 7.—N. M. was a woman, 49 years old. The preoperative diagnosis was appendical abscess with sinus. On May 7, 1941, gastrointestinal series and barium enema were given. The appendix was visualized. May 16, 1941, appendectomy was performed; histologically chronic, active appendicitis.

CASE 8.—M. C. was a 47-year-old man. The preoperative diagnosis was chronic appendicitis. June 10, 1941, gastrointestinal series and barium enema were given. The appendix was visualized and on June 13, 1941, appendectomy was done.

CASE 9.—H. K. was a woman, 59 years old. The preoperative diagnosis was chronic appendicitis. On July 23, 1941, gastrointestinal series and barium enema were given. The appendix was visualized and diverticulosis of the colon was found. July 31, 1941 appendectomy was performed and the appendix was found retrocecal, adherent, and dilated by fecaliths.

CASE 10.—J. S., a male, was 27 years old. The preoperative diagnosis was interval appendicitis. On Sept. 14, 1941, barium enema was given; the appendix was not visualized. Fluoroscopic examination was made of the stomach. Sept. 18, 1941, appendectomy was done and the appendix was found retrocecal, containing concretions.

From Oct. 1, 1940, to Sept. 30, 1941, there were 141 appendices examined histologically at the Buffalo Childrens Hospital. In the following four specimens what appeared to be barium sulfate was found microscopically.

CASE 1.—M. H., was a female, 10 months old. The preoperative diagnosis was intussusception. On Oct. 17, 1940, a barium enema was given. There was a filling defect of the cecal and ascending colon due to intussusception. On Oct. 18, 1940, there was a reduction of intussusception and appendectomy was done.

CASE 2.—B. D. was a 2-year-old girl. The preoperative diagnosis was intussusception. On March 11, 1941, a barium enema was given. There was cupping, and a filling defect in the midtransverse colon which receded to the cecum under force of solution. On March 11, 1941 there was a reduction of intussusception and removal of mesenteric lymph node; appendectomy was done.

CASE 3.—F. G. was a girl, 9 years old. The preoperative diagnosis was congenital intestinal obstruction. On Dec. 29, 1936, gastrointestinal series and barium enema were given. March 24, 1941, gastrointestinal series and barium enema were repeated; the appendix not visualized. On April 28, 1941, gastrointestinal series was given; appendix was not visualized. On May 3, 1941, anterior gastroenterostomy and appendectomy were done. The appendix dilated, contained fecalith.

CASE 4.—T. I. was a 6-year-old boy. The preoperative diagnosis was intussusception. On Sept. 1, 1941, a barium enema was given. There was a deformity of cecum. Sept. 2, 1941, after release of adhesions, appendectomy was done. In the appendix, oxyuriasis was found.

From September, 1939 to October, 1940, there were 400 appendices examined histologically at the Niagara Falls Memorial Hospital. In the following three specimens, what appeared to be barium sulfate was found microscopically.

CASE 1.—C. P. was a man, 38 years old. The preoperative diagnosis was chronic appendicitis. On April 1, 1941, a gastrointestinal series was taken. The appendix was visualized and found to be tender. May 14, 1940, appendectomy was done; the appendix contained fecalith.

CASE 2.—R. D. was a 39-year-old man. The preoperative diagnosis was chronic peptic ulcer. On Sept. 3, 1941, a gastrointestinal series was given. When the appendix was visualized, it was discovered to be somewhat enlarged at its tip and slightly tender. Sept. 9, 1940, resection of the stomach was done for ulcer and appendectomy. The appendix contained fecalith.

CASE 3.—R. E. H. was a 9-year-old boy. The preoperative diagnosis was chronic appendicitis. Sept. 12, 1940, a gastrointestinal series was given. The appendix was visualized; retrocecal, behind midascending colon. Sept. 16, 1940, appendectomy was done; the appendix dilated.

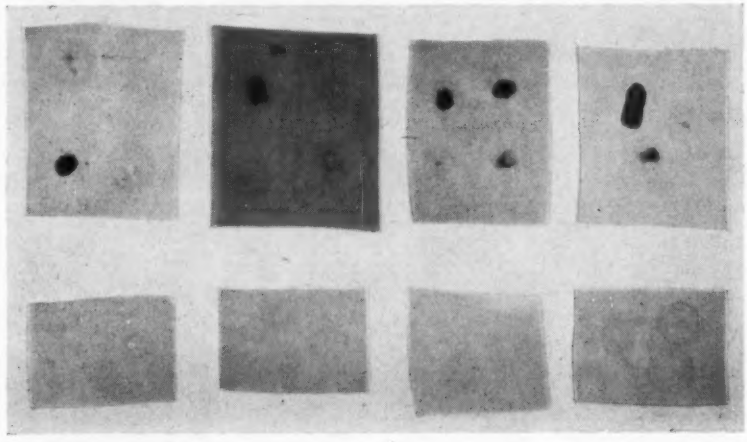

Fig. 1.—Positive prints from x-ray films of routine paraffin blocks showing, in upper row, barium in lumen of imbedded appendical segments; in lower row, control blocks with no barium in appendical lumens.

SUMMARY OF OBSERVATIONS

In 1,395 appendices examined microscopically in the laboratories of the Buffalo General, Buffalo Childrens, and Niagara Falls Memorial Hospitals during one year, what appeared to be barium sulfate was discovered in the lumen of seventeen specimens.* This discovery was made in all of the sixteen surgical cases without knowledge of history of previous radiographic examination of the patients.

Various means were employed to check our microscopic observations. In every case review of the clinical record yielded information of a gastrointestinal series or barium enema. Patients had not received oral medication which might be confused with barium sulfate. In one case

*The incidence of the microscopic diagnosis of radiopaque substance in the appendix depends on preservation of content in the preparation of sections.

a roentgenogram of the appendix obtained at autopsy verified the content to be radiopaque substance. Similar verification was demonstrated in small segments of appendix embedded in paraffin blocks from

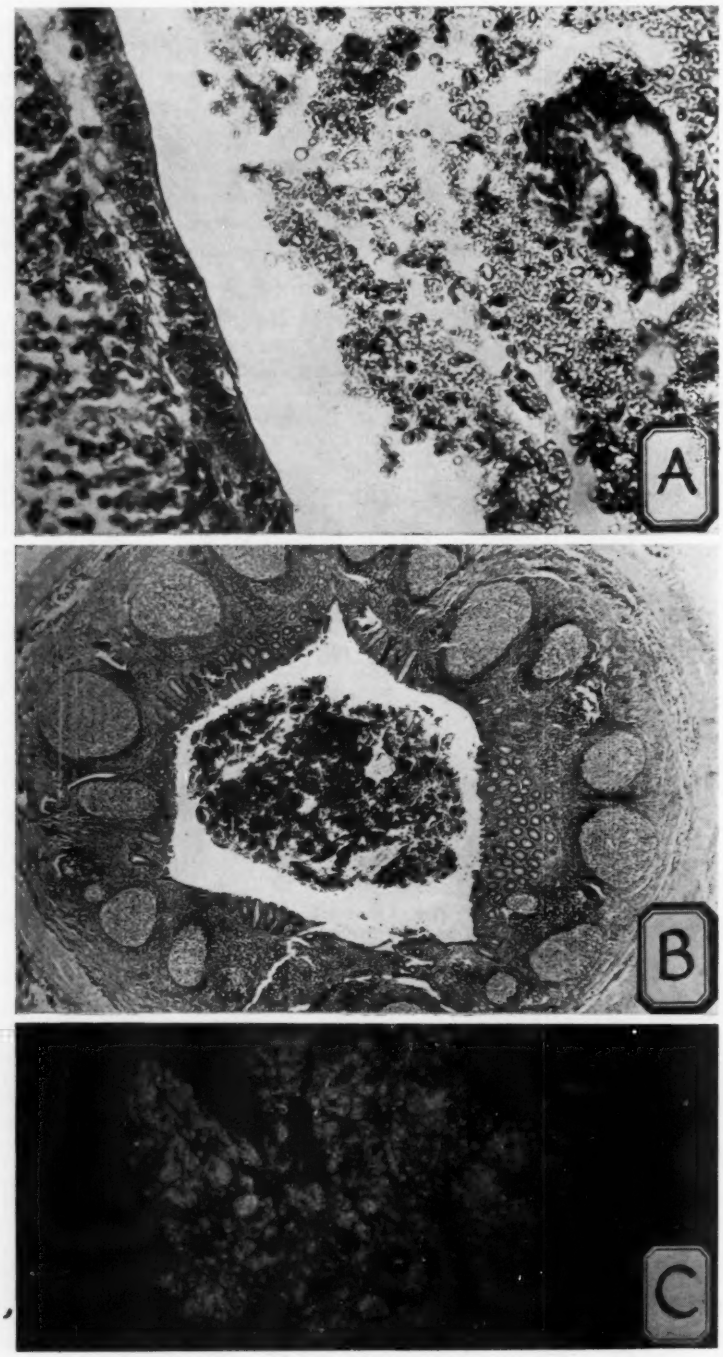

Fig. 2.—*A*, Barium sulfate particles in lumen of appendix (high power, strong central illumination). *B*, Barium sulfate particles in lumen of appendix (low power, subdued central illumination). *C*, Barium sulfate particles in lumen of appendix (low power, dark field illumination).

which sections had been cut, where these blocks were on hand. Density of radiopaque substance in paraffin blocks was differentiated from that of feces, fecalith with calcification, and foreign body. Roentgen study disclosed that if present in sufficient amount, radiopaque substances may be demonstrated in lumen of the appendix by x-ray films of slides made for histologic examination. Appendical content which was thought to be barium sulfate in sections of surgical specimens was compared microscopically and radiographically with the content of normal appendices filled with barium sulfate, bismuth subnitrate, and bismuth subcarbonate. From the last two drugs, the first was distinguished microscopically. Spectroscopic examination confirmed the presence of barium in three specimens, which were so tested.

When present in the lumen of the appendix in great amounts, material accepted to be barium sulfate was made out as a white, opaque substance in the gross specimen, in embedded segments, and in mounted sections. Rarely, barium sulfate in segments of appendix in paraffin blocks caused difficulty in cutting sections. With strong central illumination, barium sulfate appeared in sections on the microscope as glistening, green-gray, refractile, more or less amorphous material. With subdued central illumination it became darker in color. It was also observed as a glistening substance with dark field illumination either by use of ring stops with central disks, or of special reflecting condensers. In the polarizing microscope it presented no crystalline structure. The amount of barium sulfate in the lumen varied. It was distributed free in the lumen, mixed with feces, or incorporated in fecaliths.*

COMMENT ON OBSERVATIONS

At first glance the microscopic recognition of barium sulfate in the appendix might appear to serve merely as a diagnostic trick for the pathologist. By such recognition he is enabled to startle the surgeon with the report that the patient had been subjected to radiographic examination of the gastrointestinal tract before operation.

Further thought, however, on implications of the microscopic diagnosis of barium sulfate in the extirpated appendix raises a few points of practical significance.

Concerning the interpretation of certain signs in radiographic study of the appendix, there has been a difference of opinion.[3] For example, one view[3, 4] holds that the normal appendix should generally be visualized during a gastrointestinal examination. Absent or incomplete filling by radiopaque substance is looked upon as evidence of disease of the appendix. On the other side,[5] it is maintained that the "normal appendix is visualized only by the opportune coincidence of its filled condition to roentgenographic fluoroscopic examination. Nonvisualization

*As an incidental note to this appendical study, we wish to mention that we have also made the microscopic diagnosis of barium sulfate in operative and post-mortem specimens of small intestine and colon.

never necessarily means an occluded and nonfillable appendix.'' In our study we showed the presence of barium sulfate in the lumen of one appendix which had not been visualized in routine radiographic examinations on three occasions.* In respect to the problem of visualization and filling of the appendix, the microscopic diagnosis of barium sulfate along with roentgenograms of the extirpated and embedded appendix appears to offer a new and simple method, at least in one group of cases, to check the frequency with which barium sulfate enters the lumen of the appendix without detection by the roentgenologist.

Of interest is the microscopic diagnosis of barium sulfate in appendices which had not been visualized by barium enema for intussusception and which had been removed within twenty-four hours after ejection by the patient of the barium per rectum at the time of surgical reduction of intussusception.

Among the daily tasks of the pathologist is the examination of the appendix removed for ''chronic appendicitis.'' Sometimes the appendix is the site of an inflammatory process which can be related to a previous acute lesion or can be classified as a recurrent or chronic lesion. More often the surgical specimen is described histologically as ''not remarkable''; it shows no signs of active inflammation. The pathologist does not satisfactorily explain the clinical symptoms on inflammatory grounds. And the surgeon and roentgenologist interpret ''chronic appendicitis'' on a mechanical and functional basis. Wangensteen[2b] states, ''Chronic appendicitis is essentially a loosely employed clinical designation for recurrent or persistent appendical obstruction.'' From a radiographic standpoint, Holmes[4] remarks, ''In the so-called 'chronic appendix' where the infection has come and gone, there may be no evidence of the process other than fixation and failure to empty. An appendix which retains barium several days after the ascending colon has emptied is a potential source of trouble.''

In our study we noted barium sulfate which had been retained in the appendix from two to forty-three days after ingestion or enema.

Two questions arise. In retention of barium sulfate, diagnosed microscopically, do we have a direct histopathologic sign indicative of disturbance in evacuation of the appendix? How far can this sign be correlated with the clinical and radiographic picture of ''chronic appendicitis''?

We observed retention of barium sulfate in sections of appendices of 13 patients. Of these patients, 9 were operated upon for ''chronic appendicitis,'' ''interval appendicitis,'' or ''appendicular colic.'' In only 1 appendix from this group of patients was there evidence of active inflammation. The other 7 appendices revealed: fecalith and dilatation, 2; retrocecal position and adhesions, 1; fecalith, retrocecal position, and adhesions, 1; retrocecal position and fecalith or dilatations, 2; ''nothing

*Also in another appendix, without radiographic visualization, not included in this study.

remarkable,'' 2. In 4 patients with retention of barium sulfate the appendix was excised at operation, or autopsy for peptic ulcer, 2; fibroid uterus, 1; congenital intestinal obstruction, 1. From this group, 1 appendix showed partial obliteration of lumen and chronic appendicitis and periappendicitis. The other 3 revealed: fecalith and dilatation, 2; ''nothing remarkable,'' 1.

Of course, our study contains too few cases from which to draw definite conclusions on the meaning of retention of barium sulfate in the lumen of the appendix, especially of the specimen removed for ''chronic appendicitis.'' There is no clinical follow-up of our patients. We can point out that retention of barium sulfate did occur in appendices which showed active inflammation, fecaliths, retrocecal position, and adhesions. Barium sulfate, however, was also retained in 3 appendices which were adjudged ''not remarkable'' within the scope of our diagnostic criteria. This last finding might be used to support the idea that ''mere prolonged isolated filling of the appendix after the rest of the colon is free of barium cannot in itself be considered as evidence of pathologic condition.''[7] Yet, may not retention of the type under discussion still signify a disturbance of evacuation due to an impairment of motility of the appendix or ''to augmentation of normal physiologic obstruction to its emptying''[6] rather than due to obvious mechanical obstruction? On this basis, the microscopic diagnosis of barium sulfate in lumen asks for evaluation as a histopathologic sign of potential trouble in the appendix, for stasis may be a predisposing factor in appendiceal disease. It furnishes conditions favorable for development of fecaliths.

The results of our study may be considered with those reported by Wangensteen and his co-workers[2] on the detection of barium sulfate in the extirpated appendix. These investigators examined the content of appendices removed from patients who had previously received barium enema for roentgen examination of the colon, by the spectrophotometric method. Evidence of barium sulfate was absent in 13 specimens removed from 6 to 790 days after barium enema, in which the appendix was not visualized by x-ray examination, and in 4 appendices removed from 10 to 257 days after roentgen visualization with barium enema. In 6 patients with visualization of the appendix 8 hours to 6 days prior to appendectomy, barium was proved by the spectrophotometer. Morphologic descriptions of the appendix were not given in the reports of Wangensteen and his co-workers.

In 1932, Hogue[8] studied the effects of barium sulfate on tissue cultures from the chick embryo. Granules were ingested by various types of cells. In our case of chronic appendicitis with abscess and sinus, phagocytosis of granules had taken place.

We are indebted to Doctors E. C. Koenig, Ralph DeGraff, W. D. Langley, and B. R. Stephenson for roentgenographic, chemical, and spectroscopic assistance, and to Dr. K. L. Terplan for helpful criticism.

REFERENCES

1. McIntosh, J. A.: Preliminary Report on Contents of 100 Surgical Appendices, Memphis M. J. **6**: 11-14, 1929.
2. a. Wangensteen, O. H., Buirge, R. E., Dennis, C., and Ritchie, W. P.: Studies in Etiology of Acute Appendicitis; Significance of Structure and Function of Vermiform Appendix in Genesis of Appendicitis: Preliminary Report, Ann. Surg. **106**: 910-942, 1937.
   b. Portis, Sidney A.: Edited by O. H. Wangensteen, Diseases of Digestive System, Philadelphia, 1941, Lea & Febiger, pp. 730-755.
3. a. Overend, T. D.: Significance of Appendix; Radiology, Brit. J. Radiol. **10**: 829-849, 1937 (Rev.).
   b. Scholz, T.: The Solution of the Roentgen Diagnostic Problem in Chronic Appendicitis, Am. J. Roentgenol. **31**: 792-814, 1934 (Rev.).
4. Holmes, G. W., and Ruggles, H. E.: Roentgen Interpretation, Philadelphia, 1941, Lea & Febiger, pp. 300-301.
5. Larimore, J. W.: Roentgenology of the Appendix, Surg., Gynec. & Obst. **51**: 810-822, 1930.
6. Wangensteen, O. H., and Bowers, W. F.: Significance of Obstructive Factor in Genesis of Acute Appendicitis; Experimental Study, Arch. Surg. **34**: 496-526, 1937.
7. Buckstein, J.: Clinical Roentgenology of the Alimentary Tract, Philadelphia, 1940, W. B. Saunders Company, pp. 405-411.
8. Hogue, M. J.: The Reaction of Tissue-Culture Cells to Barium (x-ray) Sulphate, Anat. Rec. **54**: 307-323, 1932.

A METHOD OF RECONSTRUCTING THE SPHINCTERIC MECHANISM IN ANAL INCONTINENCE

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THE treatment of anal incontinence has received considerable attention in the past, and its surgical correction, in so far as it pertains to operative or traumatic origin, has been variously described. It is the purpose of this paper to present a new principle in anal reconstructive surgery which permits of a more favorable prognosis in those cases which have been classed as incurable or hopelessly beyond repair, and in which there are variable degrees of fecal seepage or passage of flatus.

While this condition does not compromise life, or even good health, the mental distress and the social ostracism of an individual so unfortunate as to be the victim of such a distressing condition are so disturbing that no apologies need be offered in presenting an attempt to alleviate the annoyance of such a discouraging condition.

The importance of an intact external sphincter may be more fully appreciated when it is recalled that Lockhart-Mummery stated that a person with a hopelessly cut and damaged internal anal sphincter would still have perfect control provided that the external sphincter were intact.

A new concept is here presented embodying an old principle in anal surgery, namely, that careful preliminary dilatation of the sphincters produces rest and relaxation necessary for primary healing with the minimum use of tension sutures.

The procedure consists of coaptation of the avulsed or lacerated sphincter by means of subcutaneous stay sutures, thorough, painstaking dilation and stretching, suture of the tendofascial caps, and subsequent repair of the vaginal vestibule.

ETIOLOGY

In the typical case in which this procedure is especially suitable the continuity of the external sphincter muscle has been interrupted, either as the result of anal fistula surgery or of obstetrical complications. This technique does not propose to enter into the surgical treatment of any other types of incontinence except those which are myogenic in nature; the neurogenic types, such as those resulting from neoplasms, cord

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lesions, or cerebral hemorrhage, are not amenable to these particular maneuvers, and are not within the scope of this paper.

PATHOLOGY

The resulting clinical phenomena which are manifest following the division of the external sphincter assume an inglorious role as the most distressing complication in the whole field of anorectal surgery.

The pathologic picture usually presents some degree of destruction of the perineum, and varies only in that the amount of tissue presenting itself for repair following childbirth injuries is more extensive than that encountered in incontinence following anal fistula surgery.

There is usually considerable aberration of the anatomic landmarks; the thick scar tissue which eventually invests most of the circumference of the sphincter creates a cicatricial contraction and consequent retraction of the perineal area; the retracted and contracted muscle can usually be palpated along the posterior or posterolateral aspect of the anal orifice, seldom measuring more than 2 or 3 cm. in length, and the torn ends of the muscle usually terminate in fascial and connective tissue caps firmly attached to the intervening scar tissue by dense fibrous bands where dimpling of the perineal skin is often observed.

ANATOMY

The external anal sphincter is subcutaneous and is attached, in general, anteriorly to the perineal muscles and posteriorly to the coccyx, and is divided into a superficial and a deep lamina. The muscle fibers may extend anteriorly and posteriorly on each side of the anus inserting into the sphincter vaginae, or simply into the perineal body, or a combination of the two; or, they may also encircle the anal orifice either anteriorly or posteriorly or both, and function from a relatively fixed point, such as the coccyx or central tendon of the perineum, depending upon the completeness of the anal circumference which they encompass.

It is seen, from this, that the external sphincter does not possess a fixed anatomic pattern, but that small bundles of muscle fibers find their way by diverse routes to variable fixed points in the perineum to exert their action. The blood supply to the anterior two-thirds of the sphincter and anal outlet is derived from the perineal artery, a branch of the internal pudenda which anastamoses above with the middle hemorrhoidal artery; the posterior one-third receives its blood supply from the inferior hemorrhoidal artery. The inferior hemorrhoidal nerves follow a pattern similar to that of the vascular arbor and provide innervation through each of four sectors to include the entire circumference of the anus.

PREOPERATIVE PREPARATION

We feel that, as in any other surgical procedure, proper and thorough preliminary preparation is an important step in the successful management of this problem and begin our preparation about the fourth day

when the patient is given a high colonic flushing and placed on a non-residue diet. Each day thereafter the patient is given one ounce of mineral oil and, in addition, on the evening before operation the patient is given an enema and the perineum shaved. On the morning of operation the patient is given another colonic irrigation and no breakfast, and receives, in addition, morphine and atropine hypodermically, one hour before surgery.

OPERATIVE TECHNIQUE

The perineum is cleansed with soap and water and the rectum gently irrigated. The vagina is irrigated with water and bichloride of mercury solution, and 50 to 75 c.c. of aqueous solution of mercurochrome instilled preparatory to the routine repair of the perineum which is carried out if necessary. The entire skin of the perineum is then painted with a suitable skin antiseptic.

The operation is carried out under spinal or sacral block anesthesia, or if preferred, under gas-oxygen anesthesia. At the site of the perianal dimpling a heavy catgut suture on a curved cutting needle is passed through the skin, subcutaneous tissues, fascia, and muscle. This suture is tied securely to approximate intimately the dimpled ends of the sphincter muscle (Fig. 1). The right index finger is next introduced into the rectum and with slow, gentle, firm massaging and complete anesthesia the muscle is slowly stretched (Fig. 2). Massaging should

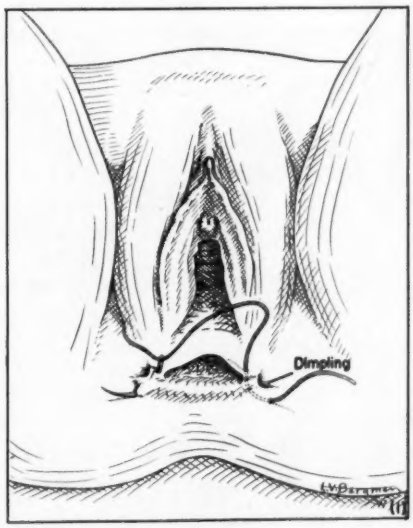

Fig. 1.

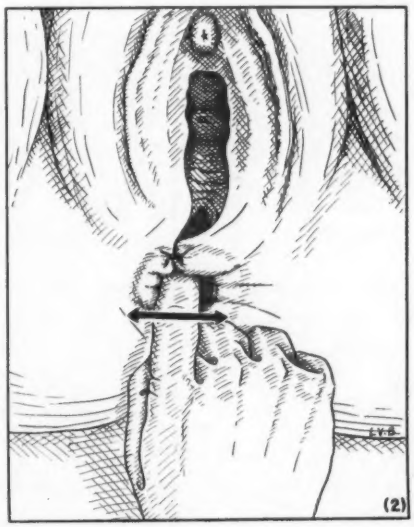

Fig. 2.

be done in a lateral manner rather than anteroposteriorly until the length of the contracted sphincter muscle measures 6 to 8 cm. This procedure should consume from twenty to thirty minutes and requires the services of at least one additional assistant. When it is felt that the muscle has been completely and thoroughly relaxed and stretched the stay suture is removed.

A suitable skin incision is next made over the external sphincter and the muscle exposed (Fig. 3). By careful, painstaking dissection the fascial connective-tissue caps on the ends of the muscle are isolated and left untouched. Two interrupted No. 1 chromic catgut sutures are passed through the muscle and tied (Fig. 4), approximating the two

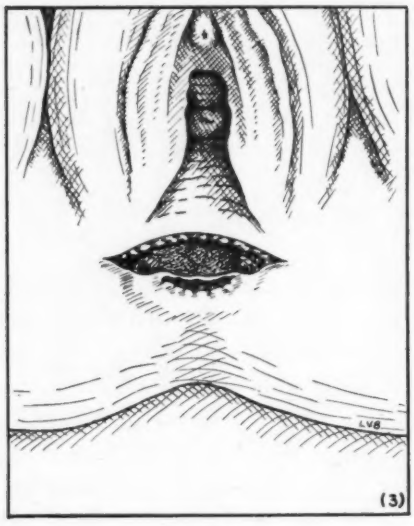

Fig. 3.

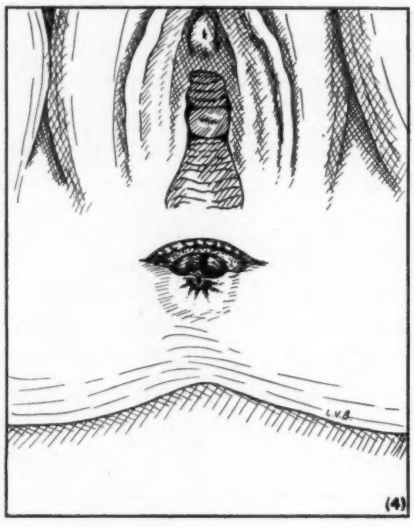

Fig. 4.

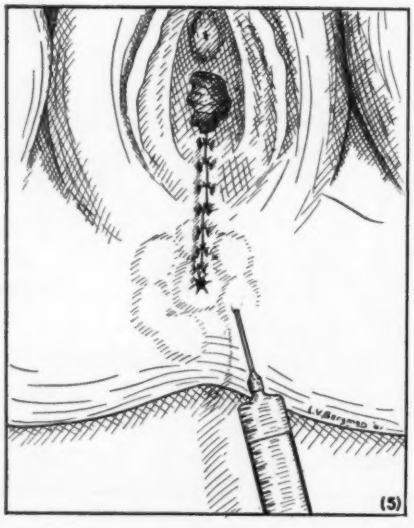

Fig. 5.

ends without strangulating the tissues involved in the suturing. The subcutaneous tissues are approximated and closed with interrupted sutures of No. 2 chromic catgut and the skin closed with black silk, silkworm-gut, or dermal sutures. If indicated, a routing perineorrhaphy is carried out.

At the close of the operation the perianal skin subcutaneous tissues and muscles are infiltrated with nupercaine in oil (Fig. 5) as a measure of relieving any postoperative pain. Bowel movements are prevented by administering ten drops of tincture of opium three times daily for eight to ten days. The patient is placed on a nonresidue diet for ten days after which time bulkier foods are added. On the tenth postoperative day the patient is given oil retention enema and one ounce of mineral oil daily, to encourage soft-formed stools.

CASE REPORTS

CASE 1.—This was a white woman, 30 years old. At the age of 20 years she had developed a fistula-in-ano which recurred after each of two operations. Sphincter control was much impaired after the second operation and completely lost after two subsequent attempts at repair. In all, twenty-eight operations were performed for fistulas. The last was successful when the patient was 26 years of age. Seven different surgeons declined to attempt surgical repair because of the apparently hopeless outlook. An attempt at repair was promised on one occasion providing the patient would first submit to a colostomy. This she refused to do.

The following significant findings were revealed on physical examination: Slight rectocele, and replacement of the perineal body with thick scar tissue. Anal orifice dilatable to admit one finger. No anal sphincter except a 2 by 2.5 cm. muscle mass posteriorly. Entire anal sphincter surrounded by cicatricial tissue, except for the small area posterior to the anus.

The operation described above was carried out with an uneventful convalescence. There was normal control of defecation and gases on the eighth postoperative day. Nine months later the patient reported good control and digital examination disclosed a normally functioning anal sphincter.

CASE 2.—A white woman, 35 years of age, presented herself with a history of recurring fistulo-in-ano of seven years' duration for which she had undergone four operations, after the second of which sphincter control was completely lost.

At 31 and 32 years of age she submitted to the last of the four procedures, without any benefit. Examination revealed a sphincter about 3 cm. in length posterior to the anal orifice. The perineum was greatly scarred, the perineal body much distorted, and a first degree rectocele presented. The outlook for clinical cure seemed hopeless. The surgical maneuvers described above were carried out and on the fourth day after operation the patient was able to expel or retain gas at will. On the twelfth day she had good control of defecation. Four years later the patient reported good control, and examination disclosed normal sphincteric function.

SUMMARY

A new technique is presented for reconstruction of the external anal sphincter in traumatic or operative incontinence. We feel that the procedure detailed above is of merit in properly selected cases of anal incontinence, and that it offers a definitely optimistic prognosis in many cases which were formerly classed as incurable.

REFERENCES

1. Hiller, Robert I.: The Anal Sphincter and the Pathogenesis of Anal Fissure and Fistula, Surg., Gynec. and Obst. **52**: 921-940, 1931.
2. Lockhart-Mummery, J. P.: The Causes and Treatment of Rectal Incontinence, Lancet **2**: 535-537, 1933.
3. Curtis, A. H.: Gynecological Diagnosis, Philadelphia, 1921, W. B. Saunders Co.

CARCINOMA METASTASES IN APPENDICES EPIPLOICAE

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ROUVIER[1] in his *Anatomy of the Human Lymphatic System* does not mention the lymph drainage in the appendices epiploicae. While studying surgically removed specimens of carcinoma of the rectum and sigmoid which had been cleared by the method described by us[2] earlier, we were surprised to find lymph nodes often present in the appendices epiploicae. These may be from 1 to 4 mm. in diameter. In some cases they are found near the base of the appendix epiploica where it first becomes a separate stalk. More often they lie 1 to 2 cm. removed from the base, or they may be found almost at the tip of the appendix. It seems to make little difference whether the appendix epiploica is thin and slender or broad and relatively massive.

In five specimens, carcinoma metastases were present in lymph nodes found in appendices epiploicae. Fig. 1 is a drawing of one of these. This surgical specimen (No. 44112) is an annular carcinoma of the middle sigmoid removed from a man 56 years of age. It was firmly adherent to the lateral abdominal wall over an area three inches in diameter. The liver was normal. There were a number of large, palpable lymph nodes in the mesentery. The rectum and sigmoid were mobilized extensively and an obstruction resection was performed after resecting a 1 cm. layer of the abdominal wall around the adherent area. He made an uneventful recovery except for a paralytic ileus which persisted for nine days. The specimen contained forty-four lymph nodes which were sectioned microscopically. One node, 3 mm. in diameter, was found in the mesentery draining the tumor. This node was completely replaced by carcinoma. A lymph node 2 mm. in diameter was found in a small appendix epiploica just at the lower end of the tumor. This node was about 0.5 cm. from the base of the appendix and it was half replaced by carcinoma. There were eight appendices epiploicae in this specimen. The fourth appendix above the tumor had a 1 mm. lymph node which had a small subcapsular metastasis. There were three appendices between the tumor and the one containing a metastasis.

The second specimen (No. 38986) was a carcinoma completely encircling the sigmoid in a woman 27 years of age. She had had symptoms for eighteen months. An obstruction resection was performed. There were lymph node metastases to four of the sixty-seven lymph nodes sectioned. Three of these nodes were in the mesentery draining the area of the tumor. There were five appendices epiploicae in the

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resected specimen. There were uninvolved lymph nodes in two appendices arising between the tumor and the one appendix containing a lymph node metastasis. This appendix was 3 cm. proximal to the tumor. The lymph node was 1 cm. lateral to the bowel wall, 2 mm. in diameter, and was completely replaced by carcinoma.

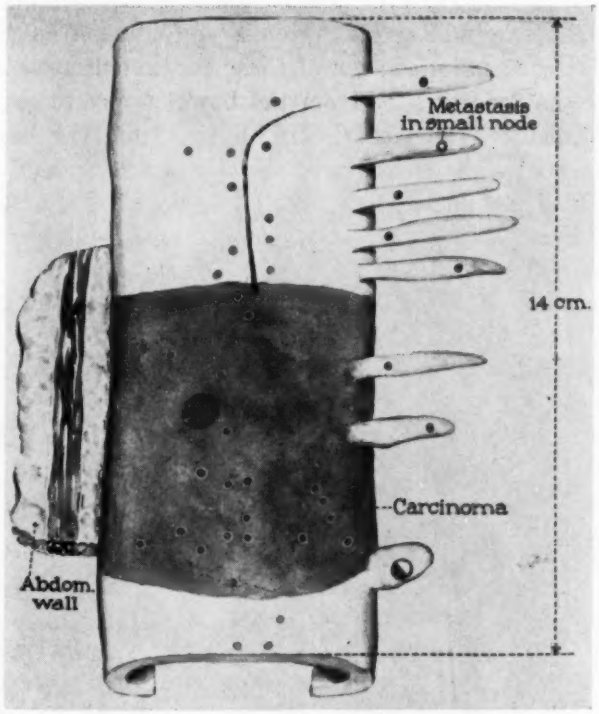

Fig. 1.—White dots over the tumor and shaded dots represent normal lymph nodes. Solid black over the tumor was a 3 mm. lymph node replaced by carcinoma. One-half of a 2 mm. lymph node in an appendix epiploica at the distal end of the tumor is replaced by a carcinoma metastasis. The lymph node 1 mm. in diameter in the fourth appendix epiploica proximal to the tumor had a small subcapsular metastasis.

The third specimen (No. 42115) was an annular carcinoma just above the peritoneal reflection. The patient was 41 years of age and had had symptoms for twenty-one months. He had developed a spontaneous fistula about 1 cm. in diameter between the carcinoma and the ileum about six inches proximal to the ileocecal valve. There were no metastases to the liver, and no peritoneal plaques. Enlarged lymph nodes were palpable to, but not above, the promontory of the sacrum. A lateral anastomosis was made around the involved ileum and a one-stage abdominoperineal resection of the rectum along with ten inches of the ileum was performed. The patient made an uneventful recovery, leaving the hospital on the twenty-second postoperative day. Ninety-two lymph nodes were microscopically sectioned in the mesentery of the rectum and seven nodes from the mesentery of the ileum. There were two appendices epiploicae in this specimen. One of these, 1.5 cm. proximal to the tumor, contained a lymph node 3 mm. in diam-

eter which was completely replaced by carcinoma. The involved node was 2 cm. lateral to the bowel wall. None of the other ninety-eight lymph nodes contained metastases. The patient was still well and working twenty months after the operation.

The fourth specimen (No. 43452) was a carcinoma involving 80 per cent of the circumference of the rectosigmoid. The patient was a 47-year-old man who had had symptoms for four months. Thirty-eight lymph nodes were sectioned; ten of these contained metastases. Three appendices epiploicae arising from the bowel opposite the tumor contained lymph nodes. One of these, 1.5 cm. from the base of an appendix, contained a 2 mm. lymph node which was half replaced by carcinoma. The patient was still well twelve months after operation.

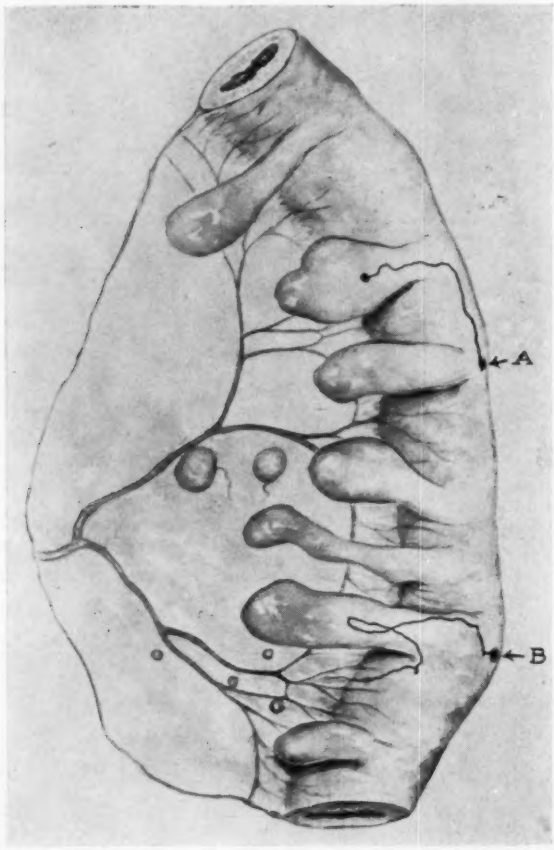

Fig. 2.—Injection of India ink suspension into antimesenteric border of human sigmoid. At *A* the lymph channel coursed in the submucosa for 2 cm. before turning laterally where it entered a lymph node 1.5 cm. from the base of the appendix epiploicae; it then made a loop and drained into a mesenteric node.

The fifth specimen (No. 40265) was an annular carcinoma of the sigmoid removed from a 61-year-old man who had had symptoms for three months. He had numerous metastases to the liver. There were several large distended diverticula above the tumor. Since the tumor could be removed easily, a palliative obstruction resection was per-

formed. Thirty lymph nodes were sectioned; four nodes contained metastases. In one appendix epiploica arising over the tumor, a lymph channel packed with carcinoma was found 1 cm. lateral to the base of the appendix.

In an attempt to reproduce the course of the metastases found in these specimens, injections of an India ink suspension were made into the subserosa of the antimesenteric border of the sigmoid of freshly performed autopsy specimens. With practice, less than 0.5 c.c. of the suspension can be injected and sometimes its course can be followed through the lymph channels. This injection must be made with very little pressure. The accompanying illustration (Fig. 2) shows one such preparation which was cleared as described.[2] At *A* the solitary lymph channel was filled with the black suspension and this passed up the antimesenteric border of the bowel in the subserosa for 2 cm. before it turned laterally and entered an appendix epiploica. The suspension filled a 1 mm. lymph node 1.5 cm. lateral to the base of the appendix epiploica. Another injection *B*, somewhat below this one, entered a single lymph channel which immediately ran into an appendix epiploica. It coursed laterally for 2.2 cm. from the base of the appendix and then made a loop and an exit from the mesenteric side of the base of the appendix and coursed down the mesentery as shown. This one preparation duplicated the manner of spread seen in our five specimens.

CONCLUSIONS

The injection preparations demonstrate that there are lymph channels in the subserosa on the antimesenteric border of the sigmoid which may travel at least 2 to 3 cm. lengthwise of the bowel before turning laterally to drain into the mesentery. If there are appendices epiploicae in this area, the lymph channel may drain into lymph nodes present in them, or the channels may simply traverse the appendix and drain into some of the mesenteric nodes. These injection specimens demonstrate the fact that at least 3 cm. of the sigmoid proximal to a carcinoma must be resected if a radical resection is to be performed. The finding of metastases in lymph nodes in appendices epiploicae 2 to 3 cm. removed from the primary tumor confirms this.

REFERENCES

1. Rouvier, H.: Anatomy of the Human Lymphatic System, Tobias Translation, Ann Arbor, Mich., 1938, Edwards Brothers, Inc., pp. 138.
2. Gilchrist, R. K., and David, V. C.: Lymphatic Spread of Carcinoma of the Rectum, Ann. Surg. **108**: 621-642, 1938.

HEMANGIOPERICYTOMA OF THE OMENTUM

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THE case recorded below seems worthy of presentation because it is an example of a rare form of vascular tumor of considerable size and astonishingly long duration and freedom from symptoms occurring in the omentum where tumors of a similar nature have not been previously described. The name was recently suggested by Stout and Murray[1] when they reported nine tumors with similar histopathologic characteristics.

The clinical history (made available by Dr. Rineamor, Miami, Fla.) reveals that for over sixty years, the patient, S. B., white, aged 92 years, noted a mass in the right para-umbilical region. Without causing any distress or symptoms other than its mere presence, the tumor had grown very slowly and gradually, reaching its greatest size, about that of a large kidney, several years before the patient's death. The mass was firm, nontender and easily movable, seemingly not attached to any viscera. No bruit was heard over the abdomen. Although the patient was aware of the mass and had felt it for about sixty years, he never had it investigated since it was perfectly symptomless throughout the patient's life. Having had hypertension, the patient began having fibrillations a few weeks before his death. Signs and symptoms of fluid in the chest appeared, and exitus came with heart failure. The patient had no genitourinary or gastrointestinal complaints.

Post-mortem examination* was limited to the abdominal cavity. About 100 c.c. of clear, straw-colored fluid were found in the peritoneal cavity. The kidneys were small and very firm. The tumor was found to the right of and just below the umbilicus. It was freely movable. The autopsy findings were otherwise noncontributory, being principally those of arteriosclerotic disease.

Intimately bound up within the greater omentum and adherent to the mesocolon was a large ovoid tumor measuring 12 cm. transversely, 9 cm. from above downward, and 8 cm. from before backward. It was nodular, encapsulated, and had some large veins coursing over its surface and apparently branching into the substance of the tumor. The consistence was firm, the color a mottled light gray.

The cut surface had a mottled appearance due to the presence of many minute and some larger vessels filled with blood. In the center of the tumor was a zone of fibrosis and necrosis. The necrotic portion had a faintly yellow tinge. There was no evidence of spread or infiltration, as the tumor was well circumscribed and no lymphadenopathy was present.

On microscopic examination this tumor was seen to be composed basically of tremendous numbers of small, and occasionally large, capillaries lined by endothelial cells and supported by extremely delicate reticulin fibers immediately external to the

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*The authors are indebted to Dr. Philipp R. Rezek, pathologist of the Jackson Memorial Hospital, Miami, Fla., for permission to study the material and publish the case.

endothelial cells. Surrounding the reticulin fibers were massed several layers of cells of varying shapes and sizes, chiefly rounded, ovoid, and spindle-shaped. These cells seemed to contain no myofibrils or acidophilic prefibrils. The capillaries in general were so closely grouped that their satellite cells came into contact one with another so that the general effect was that of a mass of these cells with the capillaries scattered through them. The true relationship of cells and capillaries was only occasionally seen where there was more fibrous tissue separating them. The larger vessels in this tumor usually had fibrous walls without satellite cells and also without smooth muscle. No elastic fibers were seen with the acid orcein stain. No mitoses were identified and the growth seemed well encapsulated.

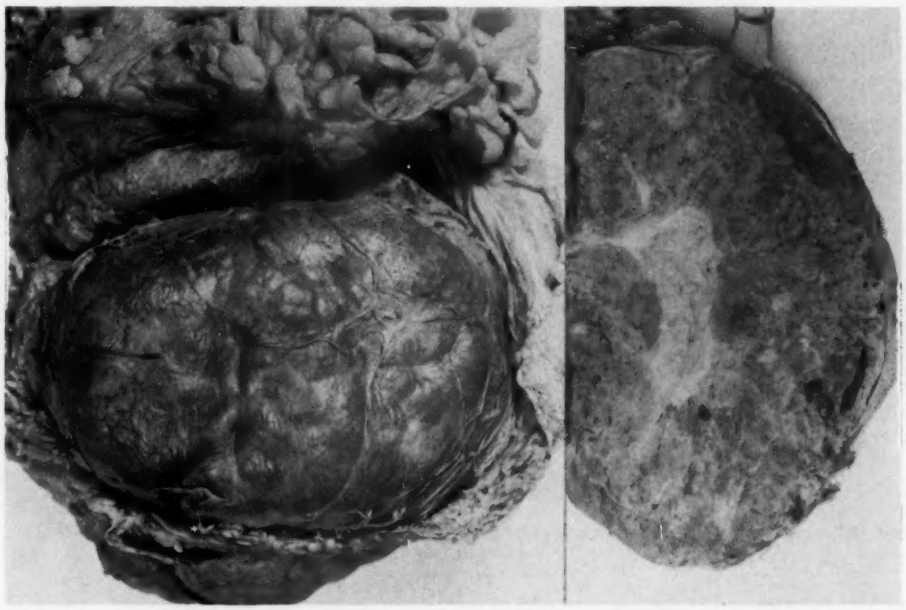

Fig. 1.—At the left the encapsulated tumor is shown wrapped about by omentum with large veins coursing over its surface. Above is the transverse colon. At the right is shown the cut surface of the tumor stippled with large and small blood vessels.

Since it seemed to be established that this was truly a tumor of blood vessels and not simply a highly vascular tumor of some other kind, the nature of the perivascular tumor cells came into question. It seemed incredible that they could be derived from the vascular endothelium which was everywhere of normal appearance and showed no evidence of unusual activity or morphologic change. The cells could be satisfactorily explained if we could believe that they were derived from Zimmermann's pericytes.[2] These are cells which are wrapped tightly about the capillaries and have contractile properties which, it is supposed, serve to narrow the caliber of the capillary lumen. Zimmermann[2] believed that they are modified smooth muscle cells. Recently Dr. Margaret R. Murray, by means of tissue culture, demonstrated that the epithelioid cells of the glomus tumor are pericytes.[3] Murray and Stout,[1] in reviewing a collection of 691 blood vessel tumors of all kinds found nine cases among them which in many ways resemble the present case. It was felt

that they did not fulfill the criteria of glomus tumors but that the cells arranged about the vessels were probably pericytes and it was suggested that the proper name for them might be hemangiopericytoma.[1] This present case has strong morphologic resemblances to the majority of these nine cases and seems to belong to the same group. A majority of the tumors pursued a benign course, but one exhibited aggressive infiltra-

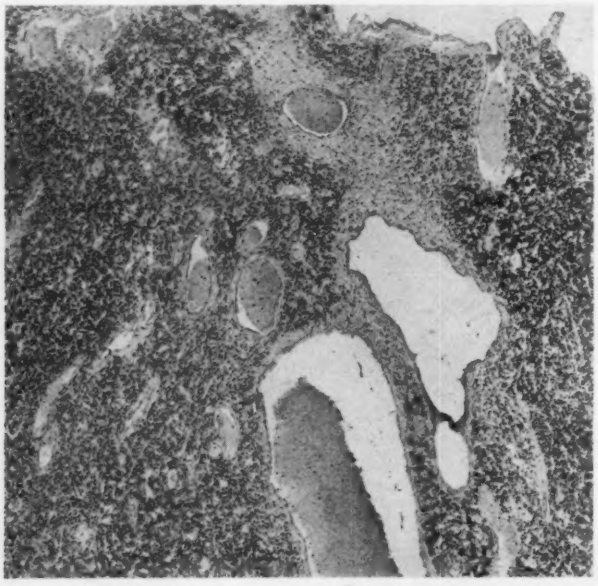

Fig. 2.—Low-power photomicrograph of the tumor showing the distribution of the vessels and tumor cells.

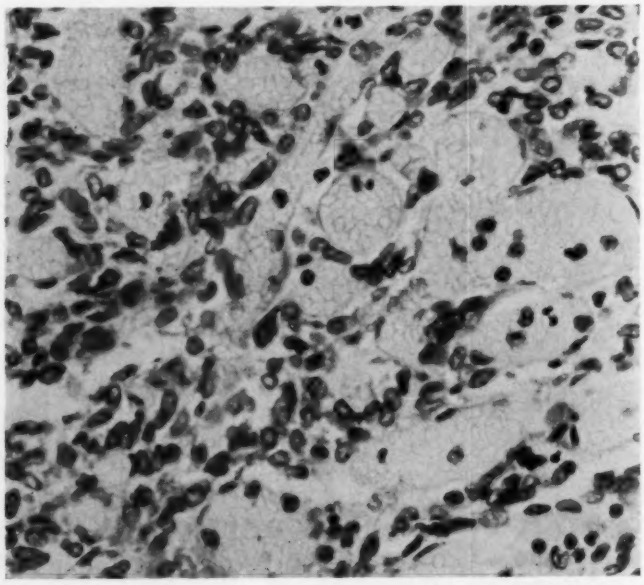

Fig. 3.—Detail showing the relationship of the tumor cells (pericytes) to the endothelial-lined capillary tubes filled with red blood cells.

tive growth which led to the amputation of the finger before it was completely eradicated, and another metastasized to the liver and caused death. The tumor in each of these nine cases was located in the parietes, cutaneously, or subcutaneously, while in the present case the site is visceral, heretofore not reported. Its size is somewhat larger than any of the previously reported similar tumors.

SUMMARY

A large encapsulated vascular tumor of an unusual form has been described which apparently arose in the omentum and was palpable by the 92-year-old patient during the last sixty years of his life. The tumor was composed of capillaries surrounded by cells believed to be proliferated Zimmermann's pericytes. For this reason the name hemangiopericytoma first suggested by Stout and Murray has been used as a descriptive term.

REFERENCES

1. Stout, A. P., and Murray, M. R.: Hemangiopericytoma (A Vascular Tumor Featuring Zimmermann's Pericytes), Ann. Surg. **116**: 26, 1942.
2. Zimmermann, K. W.: Der feinere Bau der Blutcapillaren, Ztschr. f. Anat. u. Entwcklingsgesch. **68**: 29, 1923.
3. Murray, M. R., and Stout, A. P.: The Glomus Tumor; Investigation of Its Distribution and Behavior and the Identity of Its "Epithelioid" Cell, Am. J. Path. **18**: 183, 1942.

EARLY AND LATE TOPICAL USE OF SULFATHIAZOLE IN INFECTED WOUNDS*

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THE introduction of sulfonamide drugs directly into wounds received its impetus following Jensen and associates'[1] publication in 1939. They noted superior results following the introduction of powdered sulfonamides into débrided compound fractures and primary closure. The incidence of infection was reduced from 27.2 to 4.88 per cent.

Following the article by Jensen and his co-workers, many other workers reported their results on the local use of these drugs in clean, contaminated, or infected wounds. Most investigators have commented favorably upon its local use; others claim deleterious effects, particularly in clean wounds.

Key and Burford,[2] in 1940, found a high degree of protection against staphylococci infections in compound fractures following local applications of sulfanilamide. In a subsequent article[3] they also advocated treating clean or potentially infected wounds. They reported 150 consecutive noninfected postoperative wounds treated by using a small amount of the drug in each wound.

Meleney[4] points out that surgeons usually do not appreciate the number of postoperative wound infections in a series of clean hospital cases. Careful evaluation reveals an incidence of 5 to 20 per cent, varying from mild to severe infections.

Hawking[5] found the drug diffuses quite rapidly through the wound cavity, more slowly through dead tissue, and penetrates only 2 to 3 mm. into living tissue. The basis for using sulfonamides locally is the high concentration of the drug obtainable in the immediate environment of the wound bacteria. Lockwood[6] pointed out two additional factors influencing infection and wound healing: (1) the mobilization of cellular defense over which there is no control; and (2) the chemotherapy-inhibitor substances released by enzymatic tissue dissolution which are present in larger quantities in necrotic tissue or abscesses.

Donahue[7] reported favorably on treating genitourinary wounds locally by dusting them with sulfanilamide. He concluded that local application reduced systemic reactions; the drug's solubility mitigated against its acting as a foreign body; and more wounds healed per primum. He cautioned that overdosage may cause tissue damage.

*This problem was investigated before active service in the U. S. Army.

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Reports in the English literature[8] show a marked reduction in war wound infections following routine combined oral and topical use of sulfanilamide and sulfapyridine. Moorhead's[9] recent report on treating wounded soldiers and sailors who were at Pearl Harbor indicated that the administration of local sulfonamides materially reduced wound morbidity and the mortality.

Reed and Orr[10] recently reported their comparative results on the local use of six common sulfonamides in gas gangrene infections. Sulfathiazole was found superior to sulfanilamide and other derivatives, due to its slower absorption and greater bacteriostatic action.

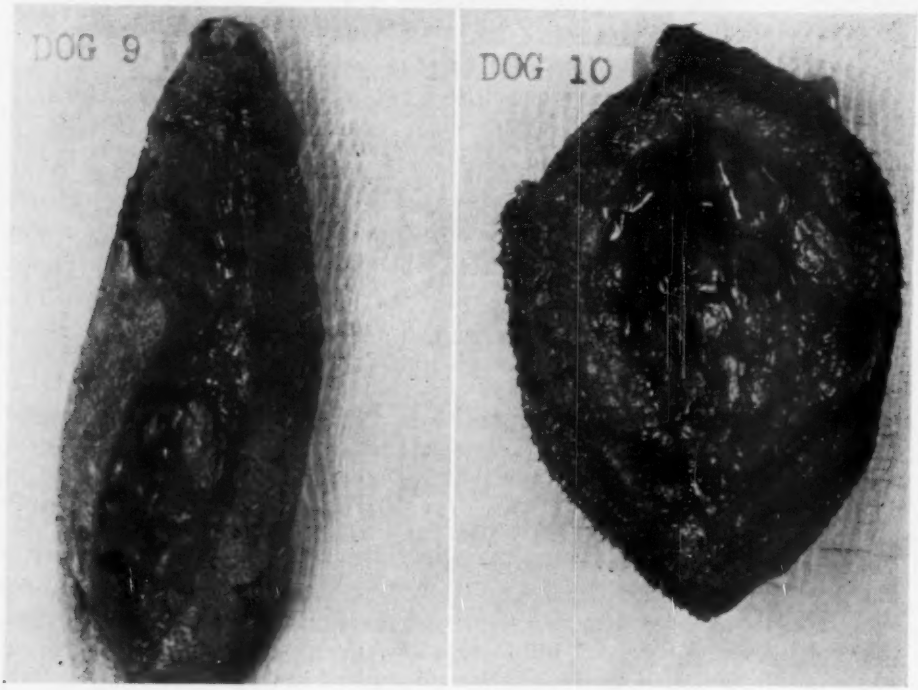
In this series of experiments the local use of sulfathiazole in contaminated and infected wounds was investigated. Extraperitoneal, renal, and low abdominal wounds were contaminated with a heavy suspension of *Staphylococcus aureus*. Powdered sulfathiazole was applied locally immediately or after two to seven days. The powder was introduced by "dumping" from the bottle with a spatula and later by using an insufflator. Three groups of adult dogs were used. The operations were performed under sodium amytal intravenous anesthesia.

GROUP 1.—Eight dogs were used; no controls. A perirenal wound was infected in six dogs with a staphylococcus suspension. Sulfathiazole powder was introduced into the wound immediately in two dogs, and after two, two, four, and eight days in four dogs. In the animals given delayed treatment, the wounds were partially or completely open and suppurating. Extension of the infection was indicated by the progressive weakness of the animal, refusal to take water and food, and febrile reactions from 2 to 4° F. The reopening of these wounds for local treatment revealed deep and superficial discoloration and induration. These reopened wounds were treated with 1 to 3 Gm. of sulfathiazole. The subsequent course revealed a control of the infection and wound healing in three to six days. In two dogs each perirenal wound was contaminated and treated immediately with 1.5 and 2 Gm. of sulfathiazole. These two wounds appeared to be healed in two days, and remained closed. In the two remaining dogs of this group, low extraperitoneal abdominal wounds were infected and treated after seven days with 1 and 2 Gm. of the drug. The wounds appeared healed in four days.

GROUP 2.—Eight dogs were used, four treated and four controls. Two dogs were operated upon during the same afternoon. One perirenal wound was contaminated with staphylococci and treated immediately with 2 to 3 Gm. of sulfathiazole. The control wound was similarly contaminated, but untreated. The wounds were all closed in layers with interrupted fine silk. Three of the four treated wounds were healed in five to seven days; one was partially open the fifth day. Two of these wounds revealed slight drainage from their dependent angle. In the control group, three wounds were open and suppurating on the fifth, fifth, and seventh postoperative day (Figs. 1 *A* and *B*). One dog died on the second day, and the post-mortem studies revealed a spreading retroperitoneal infection. The febrile reactions in the untreated dogs were 1 to 2 degrees higher than in the treated series.

GROUP 3.—Nine dogs were used. In this group bilateral lower abdominal extraperitoneal wounds were utilized. The right wounds were contaminated and treated with 1 to 3 Gm. of sulfathiazole immediately or after two to three days. The left wounds were contaminated but not treated. The treated wounds revealed better healing response than the untreated. With two exceptions, the treated wounds

revealed no discharge and healed rapidly per primum. In two treated wounds there was slight drainage after two and seven days. Both of these draining wounds healed completely in ten and sixteen days. Two treated wounds revealed a fluctuant swelling without induration. Aspiration of these two wounds revealed a reddish white fluid. Determinations on these fluids for sulfathiazole concentration revealed 94.4 and 35.3 mg. per cent. In the nine control wounds there was induration, suppuration, and drainage in seven. Two of the wounds, in spite of their marked induration, did not suppurate or drain.



A. *B.*

Fig. 1.—*A*, Partially healed perirenal wound from Group 2, Dog 9, five days after immediate treatment with 2.0 Gm. sulfathiazole had been instituted following contamination. This wound was not draining.

B, Unhealed perirenal wound from Group 2, Dog 10, five days after contamination. No sulfathiazole was applied. The wound is open widely and draining.

Microscopic Sections (Manshardt).—Wound closure was observed in all cases except those which were removed for microscopic study. Treated and untreated sets of wounds were removed for histologic studies from two to twenty-eight days postoperatively. In general there was no striking difference in the treated or untreated wound sections (Figs. 2 and 3). In early infections, treated and untreated tissues were infiltrated with leucocytes. The vessels in the vicinity were markedly hyperemic. Tissue edema was apparent where previous induration had been noted. Foreign body response was not marked in any of the wounds. After complete healing, treated and control wounds appeared alike on microscopic sections.

COMMENT

Infected wounds treated locally with powdered sulfathiazole uniformly healed more rapidly and showed less reaction than did similar untreated wounds. When sulfathiazole powder was introduced im-

mediately into a contaminated wound, bacteriostasis of the *Staph. aureus* could be accomplished. When the drug was introduced into an infected wound, the extension of the infection was arrested and reparative processes were initiated. Closure of infected sulfathiazole-treated wounds was followed immediately by general improvement of the animal.

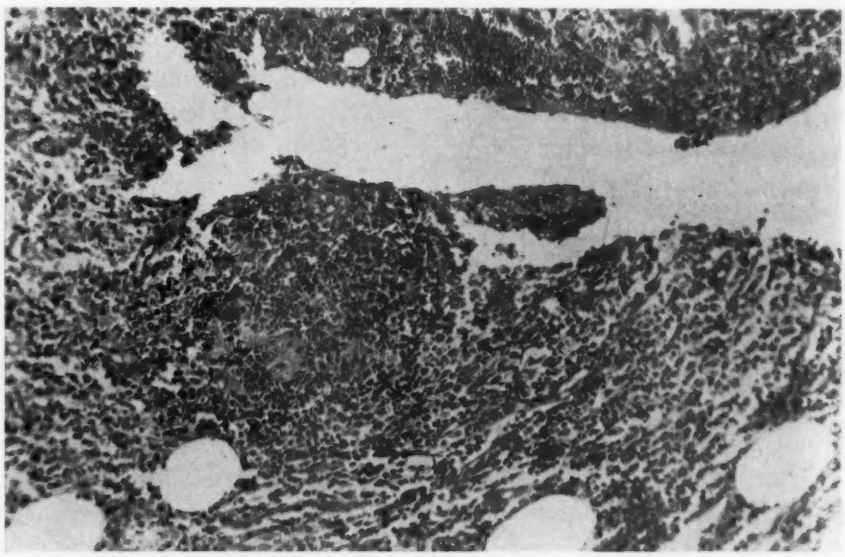


Fig. 2.—Apex of abdominal wound from Group 3 dog, seven days after immediate treatment with 2.5 Gm. sulfathiazole had been instituted following contamination. The darker staining is caused by extravasation of blood, probably traumatic.

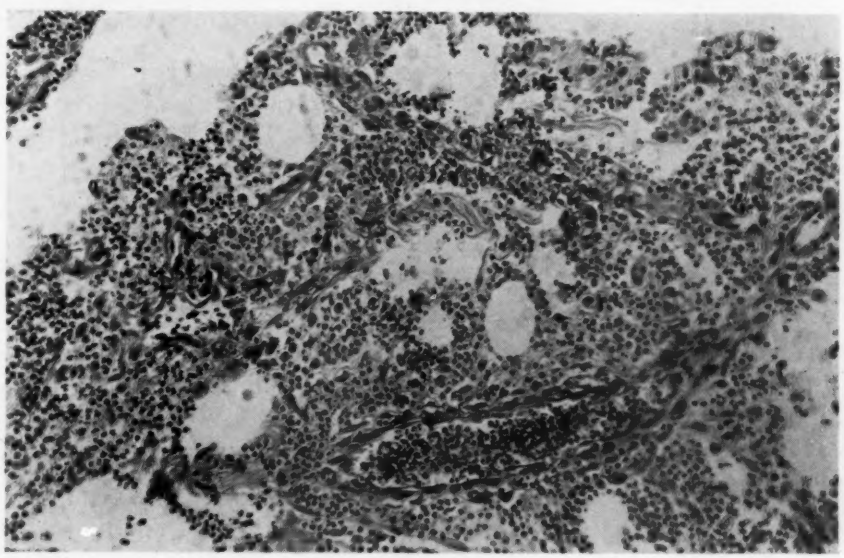


Fig. 3.—Apex of abdominal wound from Group 3 dog, seven days after contamination. No sulfathiazole was applied to this wound.

Note that both sections, Figs. 2 and 3, show apparently healthy granulation tissue. There is no significant difference in the histologic pictures.

The dosage of the drug should be determined by the size and the condition of the wound. Indiscriminate dumping of large amounts of the drug into a small wound may result in "caking." Acting thus as a foreign body, wound healing will be impaired and delayed. The ideal method of introducing the sulfonamide drugs in dry powder form into a wound locally is by the use of an atomizer or spray (Fig. 4). With this technique large wounds may be covered with a thin coating of the drug, including the deepest recesses. The use of moderate doses in these wounds failed to elevate the blood sulfathiazole concentration above 1 mg. per 100 c.c. of blood. Reinforced oral administration of the drug was not carried out. This is a preliminary report. Further investigations are to follow.

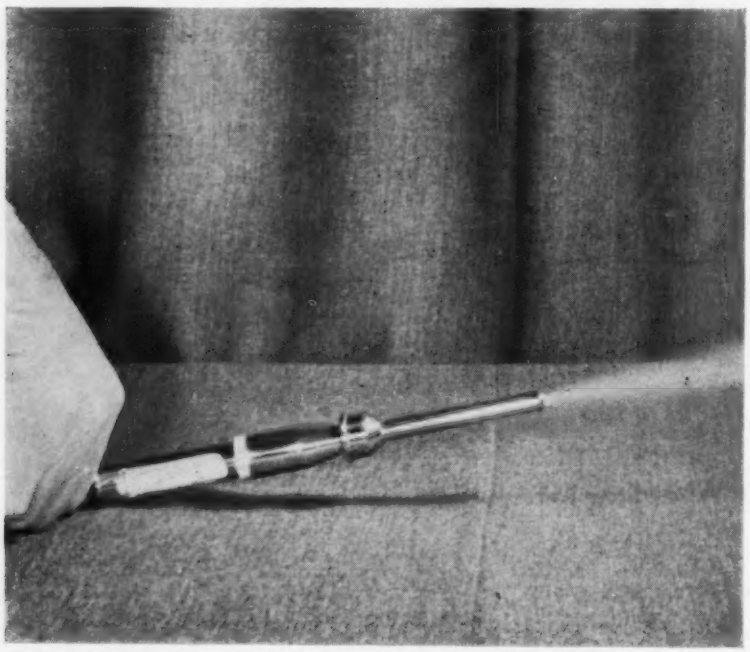


Fig. 4.—This insufflator was used for topical wound treatment. The fine cloud of sulfathiazole crystals insures complete wound coverage with small amounts of the drug.

SUMMARY

1. Healing time and extension of infection were uniformly reduced by introducing powdered sulfathiazole locally into contaminated or infected staphylococcic wounds.

2. Untoward tissue reaction to the sulfathiazole following moderate dosages was not observed in the microscopic sections.

3. Sufficient drug should be introduced to cover the wound surfaces.

4. The spray method is recommended to insure adequate wound distribution and to minimize the danger of overdosage and "caking" in the wound.

The able assistance of student John C. Jones is gratefully acknowledged. Photographic assistance was rendered by Miss Avis Gregersen and Hamilton P. Simonds.

Sulfathiazole for this investigation was furnished by Abbott Laboratories, North Chicago, Ill.

REFERENCES

1. Jensen, N. K., Johnsrud, L. W., and Nelson, M. C.: Local Implantation of Sulfanilamide in Compound Fractures, SURGERY **6**: 1-12, 1939.
2. Key, A. J., and Burford, T. H.: The Local Implantation of Sulfanilamide in Compound Fractures—Its Effects on Healing, South M. J. **3**: 449, 1940.
3. Key, A. J., and Burford, T. H.: The Prophylactic Implantation of Sulfanilamide in Clean Operative Wounds for the Reduction of Post-Operative Infections, Surg., Gynec. & Obst. **73**: 324-332, 1941.
4. Meleney, F. L.: The Relationship of Bacteriology to Surgery, Christopher's Textbook of Surgery, Philadelphia, 1940, W. B. Saunders Company, Pp. 13-15.
5. Hawking, F.: Local Concentration of Sulfonamide Compounds Inserted into Wounds, Lancet **1**: 786, 1941.
6. Lockwood, J. S.: Sulfanilamide and Its Derivatives in Surgical Infections, SURGERY **10**: 493-503, 1941.
7. Donahue, C. D.: The Use of Powdered Sulfanilamide in Infected Genito-Urinary Wounds, J. Urol. **46**: 562-566, 1941.
8. Long, P. H.: Clinical Use of Sulfonamide Compounds in Prophylaxis and Treatment of Infections, Northwest Med. **40**: 311-321, 1941.
9. Moorhead, J. J.: Surgical Experience at Pearl Harbor, J. A. M. A. **118**: 712-714, 1942.
10. Reed, G. B., and Orr, J. H.: Local Chemotherapy of Experimental Gas Gangrene, War Med. **1**: 59-78, 1942.
11. Key, J. A., and Frankel, C. J.: The Local Use of Sulfanilamide, Sulfapyridine, and Sulfamethylthiazole, Ann. Surg. **113**: 284-297, 1941.
12. Hawking, F., and Piercy, J. E.: Blood Concentrations Following Local Application of Sulphonamide Compounds to Wounds, Brit. M. J. **1**: 511-512, 1941.
13. Goodwin, L. G., and Findlay, G. M.: Absorption and Excretion of Sulfonamides Applied Locally, Lancet **1**: 691, 1941.
14. Johnson, M., Jr., and Davis, F. F.: The Use of Sulfanilamide Powder in Open Wounds, Virginia M. Monthly **67**: 748-750, 1940.

CHEMOTHERAPY IN PENETRATING ABDOMINAL GUNSHOT WOUNDS

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ALMOST from the time of their introduction as effective bacterial therapeutic agents, the sulfonamides have been used in the treatment of bacterial peritonitis. In February, 1937, I used prontosil and prontylin, intramuscularly and orally, successfully in a case of generalized hemolytic streptococcal peritonitis that followed the probable extralumenary rupture of an intramural abscess, resulting from an appendectomy in a girl 17 years of age. Other clinicians[1, 2] reported recovery of patients ill with "primary" hemolytic streptococcal peritonitis, who were treated with prontosil and sulfanilamide. In 1939, Long and Bliss,[3] in addition to the oral and parenteral administration of sulfanilamide, suggested its use in solution through a draining sinus, into the abdominal cavity, following surgical drainage. Mueller[4] reported using powered "crystalline sulfanilamide directly in the peritoneal cavity at the time of operation." This was followed by an additional report with Thompson.[5] Jackson and Coller[6] reported on "experimental and clinical observations" on the use of "Sulfanilamide in the Peritoneum." Rippy[7] used sulfanilamide intraperitoneally in treating gunshot wounds of the abdomen. Ravdin, Rhoads, and Lockwood[8] found sulfanilamide therapy to influence favorably the peritonitis of appendicitis, which is usually a mixed infection. Rosenburg and Wall[9] placed sulfanilamide into the peritoneal cavity at the close of operations for perforated appendicitis or inflammatory disease of the pelvis. Wangensteen[10] reported implanting sulfanilamide about the anastomoses in certain gastric resection cases and following resections of the colon. Following the Pearl Harbor attack, Moorhead[11] reported a high mortality among the intra-abdominal injury cases in spite of the free use of the sulfonamides. In a later report from this same source, Ravdin and Long[12] stated that in the victims of abdominal injuries crystalline sulfanilamide was of great benefit to those patients who were operated upon.

MORTALITY IN PENETRATING ABDOMINAL GUNSHOT WOUNDS

Although there appears to be a definite tendency to improve, the mortality rate in penetrating abdominal gunshot injuries is, nevertheless, still very high. The 30 per cent mortality reported by McGuire[13] and the 40 per cent reported by Storck,[14] in 35 personal cases, are

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exceptional and unusual. Reports of large series, both by civilian and military surgeons, tend to indicate that the mortality rate in these victims is usually much higher. Thus, Wallace,[15] reporting from World War I, gives a mortality rate which averages 56.5 per cent. In a study of 112 cases,[16] I found a death rate of 60.7 per cent. Oberhelman and LeCount[17] reported 222 deaths in 343 cases or a mortality of 64.7 per cent; and Rippy[7] a rate of 63.4 per cent in 369 cases. Jolly,[18] reporting from the late Spanish Civil War, gives a mortality rate of 49.4 per cent in 243 consecutive cases. I found, in a recent study of 478 additional cases,[19] a fatal result in 54.8 per cent.

CAUSES OF DEATH

There is probably no other type of injury that presents as varied a pathologic picture at operation as is frequently found in penetrating abdominal gunshot wounds, and practically all writers, reporting studies of large series of such injuries, agree that the commonest causes of death following these injuries are (1) hemorrhage and shock and (2) general peritonitis. Other causes of death, such as pneumonia, pulmonary embolism, residual abscesses, and intestinal obstruction, occur relatively infrequently. In a previous report[20] I found that of 68 deaths 54.4 per cent resulted from hemorrhage and shock, 38.2 per cent from general peritonitis, and only 7.4 per cent of all fatalities were attributed to such miscellaneous causes as previously named. Hence, 92.6 per cent of all deaths in that particular group were the result of these two causes. In the 478 cases recently studied, 56.8 per cent of the patients died of hemorrhage and shock, and 40.1 per cent of general peritonitis. Oberhelman and LeCount[17] found, in the 205 cases autopsied, that 101, or 49.2 per cent of the patients died of hemorrhage and shock, and 95, or 46.2 per cent, of general peritonitis. Rippy[21] found that "shock and hemorrhage" accounted for 59.8 per cent of the deaths, and peritonitis for 29.6 per cent.

MANAGEMENT

The management of hemorrhage and shock in these cases no doubt ranks in importance with an intelligently planned and rapidly but carefully performed operation that is actually twofold in its purpose—first, and most important, the securing of bleeding points, and second, the repair of injuries to the hollow viscera. Because of the importance of the above facts every effort is usually made to use transfusions of blood or plasma in order to overcome the ill effects resulting from the loss of large quantities of blood. Among other measures used, the recent report of Helfrich, Cassels, and Cole,[22] suggests that adrenal cortical extract may be very useful in the prophylaxis and treatment of shock.

After the control of hemorrhage and shock there is the danger of a generalized peritonitis. Application of the principles advocated by

Ochsner, Gage, and Cutting,[23] Wangensteen and Paine,[24] and Bartlett[25] has diminished the severity of many peritoneal infections, made other patients more comfortable, and has undoubtedly improved the mortality in these cases very materially. Accordingly, the use of Fowler's position, heat to the abdomen, the maintenance of an adequate fluid and electrolyte balance, the management of ileus, and the use of continuous gastric and intestinal suction drainage have been of inestimable value in the treatment of peritonitis.

The use of chemotherapy in the treatment of this condition has been very briefly reviewed in the introduction of this presentation. The beneficial influence of the sulfonamides in the peritonitis resulting from abdominal gunshot injuries has lately aroused my interest. While engaged in the study of 478 such cases, admitted to the New Orleans Charity Hospital over a period of fifteen years prior to January 1, 1942, it was noticed that in those patients receiving some form of chemotherapy the mortality rate was lower. Accordingly, it was decided to make a separate study of all such patients and to compare them with other patients admitted during the same period of time who had not received any chemotherapy during their stay in the hospital.

GROUP RECEIVING CHEMOTHERAPY

The first patient in this series to be treated additionally with a sulfonamide was admitted on July 9, 1938, and died five days later of a generalized peritonitis. This patient is the first of the 9 fatal cases briefly outlined. Since this first case, and until July 9, 1942, 48 additional patients have received chemotherapy by one or more of the usual routes. In this group of 49 cases there were 9 deaths giving the almost unbelievable mortality rate of 18.4 per cent. As none of the patients in the 9 fatal cases died within twenty-four hours of admission to the hospital, it was considered unlikely that any succumbed actually to hemorrhage and shock.

CHEMOTHERAPY GROUP I

Briefs of Fatal Cases

Hosp. No. 54321.—A white male, 37 years of age, had eleven perforations of the ileum with moderate hemorrhage. He was given prontosil, 20 c.c., every four hours. Five days after admission, he died of general peritonitis.

Hosp. No. 73224.—A negro male, 13 years of age, had two perforations of the stomach and moderate hemorrhage. There was evisceration nineteen days after operation and the patient died. He had been given 30 gr. sulfanilamide every three hours for three doses after operation.

Hosp. No. 77994.—A white male, 36 years of age had no operation. He was given 10 c.c. prontosil every four hours. Autopsy showed perforations of the liver, stomach, and right hemothorax. There was a massive hemorrhage. In forty-eight hours, he died of "hemorrhage and shock and right hemothorax."

Hosp. No. 51879.—The patient was a white male, 28 years old. Multiple intestinal perforations had been missed at operation. There was moderate hemorrhage. He was given sulfapyridine, 15 gr., every two hours for ten doses and neoprontosil, 5 c.c. "alternately" every four hours. He died of general peritonitis.

Hosp. No. 10878.—This was a negro male, 20 years of age, with perforations of the stomach, transverse colon, and ileum. Massive hemorrhage was present. Sulfanilamide was given by mouth. There was evisceration ten days after operation. The patient died of "general peritonitis and pneumonia."

Hosp. No. 42232.—A negro male, 27 years old, had perforations of the descending colon and massive hemorrhage. He was given sulfanilamide, 0.8 per cent solution (250 c.c.) by hypodermoclysis, every six hours for six doses. He died of general peritonitis.

Hosp. No. 44254.—A negro male, 37 years of age, had perforations of the ileum, jejunum, and transverse colon. Massive hemorrhage was present. He was given sulfapyridine, 30 gr., every four hours (apparently for pneumonia). Death was attributed to "general peritonitis and pneumonia."

Hosp. No. 61059.—The patient was a negro male, 40 years old, with perforations of the stomach, jejunum, and caecum. He had massive hemorrhage and was given sulfanilamide by mouth. There was evisceration ten days after operation. He died of "general peritonitis and pneumonia."

Hosp. No. 57015.—A white male, 29 years of age, had perforation of the ileum (missed at operation) and perforation of the right femoral vein and artery (both ligated). There was massive hemorrhage. Sulfanilamide was used in the operative abdominal wall wound only. He died of general peritonitis.

GROUP NOT RECEIVING CHEMOTHERAPY

During the same period of time, 51 additional patients failed to receive chemotherapy in any form. In this group there were 26 deaths, resulting in a mortality rate of 50.9 per cent. A closer study of these cases shows that 11 died within twenty-four hours of admission, and in each of these the cause of death was given as "hemorrhage and shock." Exclusion of these 11 cases would show 15 deaths among 40 cases, thus giving a mortality of 37.5 per cent.

INFLUENCE OF SULFONAMIDES ON THE VARIOUS HEMORRHAGE GROUPS

In a previous communication[20] I classified hemorrhage observed in penetrating abdominal gunshot wounds into (1) slight, (2) moderate, (3) massive, and (4) unknown. The influences on the mortality rate in the various groups are given in that paper and have been recently confirmed in the 478 cases additionally studied. A study of the influence of the sulfonamides on these various groups among the 100 cases, upon which the present paper is based, revealed some interesting data, particularly in the "moderate" and the "massive" hemorrhage groups. In both those cases in which the patients were treated with the sulfonamides and those not so treated, the mortalities in the "slight" and "unknown" hemorrhage groups were the same, as not a death was recorded in either of these groups. However, a comparison of the "moderate" and "massive" groups in each instance revealed a marked difference in their respective mortality rates. In those cases in which sulfonamide therapy was used the mortality was 12 per cent and 40 per cent respectively in the "moderate" groups, while in the cases in which no sulfonamide treatment was given the comparative rates were 30.7 per cent and 91.6 per cent, respectively. Although the number of

cases in this portion of the study is relatively small the marked differences observed appear significant and probably represent a true picture of the facts involved. The loss of blood invariably results in a diminished resistance; that is, the more blood lost the more diminished is the resistance. Accordingly, those patients losing "moderate" or "massive" amounts of blood and recovering from hemorrhage and shock, frequently succumbed to the peritoneal infection. It is this particular group which is apparently greatly benefited by the use of sulfonamide therapy, especially the intraperitoneal use of sulfathiazole and sulfanilamide. A glance at Tables I and II will undoubtedly make these points clearer and even more impressive.

TABLE I

RELATIONSHIP OF CHEMOTHERAPY TO HEMORRHAGE, IN 49 PATIENTS RECEIVING SULFONAMIDES*

TYPE OF HEMORRHAGE	NUMBER	LIVED	DIED	MORTALITY RATE (PER CENT)
Slight	8	8	0	00.0
Moderate	25	22	3	12.0
Massive	15	9	6	40.0
Unknown	1	1	0	00.0
Total	49	40	9	18.4

*In the recent group of 100 patients admitted to the New Orleans Charity Hospital from July 9, 1938, to July 9, 1942.

TABLE II

RELATIONSHIP OF HEMORRHAGE TO MORTALITY RATE, IN 51 PATIENTS RECEIVING NO CHEMOTHERAPY*

TYPE OF HEMORRHAGE	NUMBER	LIVED	DIED	MORTALITY RATE (PER CENT)
Slight	9	9	0	00.0
Moderate	13	9	4	30.7
Massive	24	2	22	91.6
Unknown	5	5	0	00.0
Total	51	25	26	50.9

*In the recent group of 100 patients admitted to the New Orleans Charity Hospital from July 9, 1938, to July 9, 1942.

This brief study would appear to indicate that chemotherapy is of distinct benefit in the treatment of these usually serious cases, after the dangers from hemorrhage and shock have passed. The importance of this study, despite the relative paucity of cases permitting more definite conclusions, is nevertheless enhanced by the fortunate availability of a control group receiving no chemotherapy and being treated, during the same period of time, as those additionally treated with the sulfonamides. (Fig. 1.)

The sulfonamide drugs used in the 49 cases, herein considered, were given orally, intramuscularly, subcutaneously, intravenously, and intraperitoneally. Since very few surgeons handled more than one case in the entire series, there is a conspicuous lack of uniformity in the use of these drugs until the method of peritoneal implantation was started.

INTRAPERITONEAL CHEMOTHERAPY

The recent report of Harbison and Key[26] in this respect seems to be of much interest. Eleven patients in the present series were treated by placing from 4 to 15 Gm. of sulfathiazole in the peritoneal cavity. Each of these 11 patients recovered, and a brief synopsis of each case is herein given. A review of the cases will show the patients to have usually suffered multiple perforations of the hollow viscera in addition to other injuries.

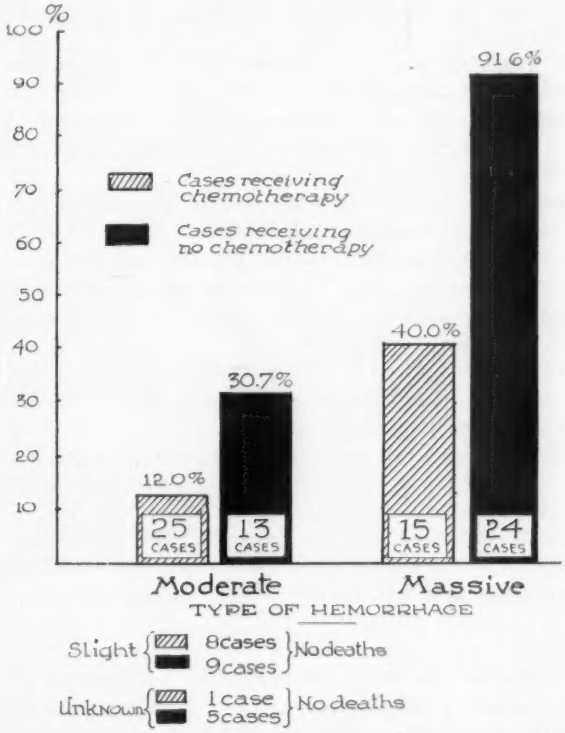

Fig. 1.—Graph showing the relationship of hemorrhage to mortality rate.

A lowered mortality rate in these cases, which was previously considered unlikely, now seems to be very possible. Although 49 cases is a relatively small number upon which to draw conclusions, this study appears to point to a more hopeful future for victims of such injuries, especially in the group succumbing to generalized peritonitis. The 11 cases in which the patients received intraperitoneal implantation of sulfathiazole are particularly impressive since in each the peritoneum was definitely contaminated, and in some, fecal matter was removed from the cavity. It would appear from these 11 cases that the intraperitoneal use of large amounts of sulfathiazole is very effective and from all indications safe and nontoxic. However, many observers of intraperitoneal chemotherapy are of the opinion that sulfanilamide is just as effective and probably less dangerous.[27, 28]

CHEMOTHERAPY GROUP II

Briefs on Cases Receiving Sulfonamide Therapy Intraperitoneally

Hosp. No. 9528.—A negro male, 15 years of age, had eight perforations of the ileum and moderate hemorrhage. He was given 10 Gm. sulfathiazole in the peritoneal cavity and recovery followed.

Hosp. No. 12244.—The patient was a negro male, 22 years of age. He had several perforations of the stomach and slight hemorrhage. Sulfathiazole, 10 Gm. intraperitoneally, was given and recovery followed.

Hosp. No. 19340.—A negro male, 38 years old, had perforations of the stomach and small intestine. There was moderate hemorrhage. Sulfathiazole, 10 Gm. intraperitoneally, was administered, followed by recovery.

Hosp. No. 26544.—This was a negro male, 30 years of age, with numerous perforations of the ileum, jejunum, and sigmoid. He had moderate hemorrhage. Sulfathiazole was given 15 Gm. intraperitoneally. The patient recovered.

Hosp. No. 1585.—A negro male, 13 years old, had perforations of the stomach and pancreas. There was slight hemorrhage. Sulfathiazole, 10 Gm. intraperitoneally, was given, and the patient recovered.

Hosp. No. 1885.—The patient was a negro male, 24 years of age, who had perforations of the jejunum and descending colon. There was moderate hemorrhage. Sulfathiazole was given, 5 Gm. intraperitoneally, and recovery followed.

Hosp. No. 11635.—A white male, 14 years of age, had perforations of the ileum and cecum. Slight hemorrhage was present. Sulfathiazole, 10 Gm., intraperitoneally, was administered. The patient recovered.

Hosp. No. 17777.—A negro male, 23 years old, had perforations of the ileum and urinary bladder. There was moderate hemorrhage. Sulfathiazole was given 5 Gm. intraperitoneally, followed by recovery.

Hosp. No. 33771.—This was a negro male, 32 years of age. He had perforations in the transverse colon, jejunum, and left kidney. Moderate hemorrhage was present. Sulfathiazole, 5 Gm. intraperitoneally was given. The patient recovered.

Hosp. No. 45847.—A negro male, 69 years old, had perforations of the jejunum and descending colon. There was moderate hemorrhage. Sulfathiazole was given, 4 Gm. intraperitoneally, and 2½ Gm. in 500 c.c. normal saline as hypodermoclysis every eight hours. Recovery followed.

Hosp. No. 47623.—This patient was a white girl, 14 years of age. She had multiple perforations of the ileum and moderate hemorrhage. Sulfathiazole was placed about the areas of bowel perforations. A sulfanilamide-sulfadiazine mixture was used in the abdominal wall wound. Sodium sulfadiazine was given, 15 gr. intravenously, every six hours and eschatin, 2 c.c., every 2 hours. The patient was operated upon forty-one and one-half hours after being shot; she recovered.

TOXICITY

In no case in this series in which the patient received chemotherapy was there any indication of toxicity from the drugs used. At least in none of the records was there any mention of cyanosis, nausea and vomiting, nor jaundice, certainly none that could be attributed to the use of the drugs.

BLOOD LEVELS

No routine blood sulfonamide levels were determined in these cases. As a matter of fact, blood levels were determined in only 9 of the 49 cases. The highest recorded was 9.2 mg. per 100 c.c. of blood, observed in Case No. 17777, in which the patient received 5 Gm. of sulfathiazole intraperitoneally. Lower levels than this were the rule in nearly all

the other cases. No concentration determinations were recorded, and none were made, on the peritoneal fluid. The records additionally indicate that none of the 11 patients thus treated was surgically drained.

SUMMARY

The introduction of the sulfonamides, in the treatment of bacterial peritoneal infections, has added what appears to be several very effective, and apparently relatively safe, drugs to our armamentarium for the treatment of this frequently fatal disease. The implantation of these drugs, particularly sulfanilamide and sulfathiazole, into the peritoneal cavity, at the completion of a celiotomy, seems to be an especially valuable and effective way of treating generalized peritonitis.

Although hemorrhage and shock usually account for more than one-half the fatalities among victims of penetrating gunshot wounds of the abdomen, general peritonitis is not far behind, and is itself a very potent factor in the fatal cases. Only a relatively small and almost insignificant number of victims of these injuries succumb to miscellaneous causes. The recent practice of using the sulfonamides in these abdominal gunshot injuries, as apparent from the study of the cases in this presentation, has resulted in an almost unbelievable drop in the mortality rate in these victims. From all indications it is quite possible that the use of these drugs frequently helps those patients who have survived a severe hemorrhage fight off the ravages of an otherwise fatal infection. Such patients, aided with large transfusions, make unbelievably rapid recoveries.

Despite the fact that some opinions have been rather forcefully expressed regarding the dangers of the toxicity of these drugs, particularly when introduced intraperitoneally in large doses, their use in the treatment of penetrating abdominal gunshot injuries appears to be especially indicated. Although some surgeons have felt, in using the sulfonamides intraperitoneally, that the dangers of the drugs are greater than the risks of the infection, I am of the opinion that this feeling should not prevail when penetrating gunshot wounds of the abdomen, with perforations and lacerations of the hollow viscera, are considered. Certainly in the group of 49 cases herein considered there was no record of any toxic symptom attributable to these drugs.

Routine blood sulfonamide levels should be run on all cases treated with these drugs, especially when large doses are used. In cases with peritoneal drainage, a study of the peritoneal fluid concentrations, when compared with the blood levels, will at least give some idea of the rate of absorption of the drug. Among those cases in which intraperitoneal implantations of sulfathiazole were used, the amount of the drug used was no criterion to the blood level expected. Some patients receiving relatively large amounts intraperitoneally showed relatively low blood levels and vice versa.

The intraperitoneal use of the sulfonamides has appeared to obviate the need for drainage in these cases. Removal of gross contaminations

with suction or otherwise seems to suffice if the drug is going to be placed, in adequate dosage, into the peritoneal cavity. Additional administrations of these drugs may be by any of the several routes otherwise available to the surgeon.

CONCLUSIONS

1. A brief review of chemotherapy in general peritonitis is presented, showing the favorable influence of the sulfonamides upon this dreaded disease.

2. The mortality rate in penetrating abdominal gunshot wounds is still frightfully high, although a tendency to improvement appears evident.

3. About 90 per cent, and usually more, of patients with penetrating abdominal gunshot injuries succumb to (a) hemorrhage and shock, or (b) generalized peritonitis. The former cause usually predominates in large series of cases, although general peritonitis is practically invariably a very close second.

4. The management of these victims comprises the treatment of hemorrhage and shock, and, the treatment of peritonitis, which is practically always a complication when hollow viscera are injured.

5. A group of 100 cases of penetrating gunshot wounds of the abdomen admitted to the New Orleans Charity Hospital from July 9, 1938, to July 9, 1942, constitutes the material upon which this study is based. Forty-nine received some form of chemotherapy, whereas 51 were more or less similarly handled except that no sulfonamides were used as additional treatment.

6. In the chemotherapeutically treated group the mortality rate was 18.4 per cent. The 51 patients receiving no sulfonamide treatment showed a mortality of 50.9 per cent.

7. A closer study of the group treated with the sulfonamides is very impressive. The 11 patients getting intraperitoneal implantations of sulfathiazole, and showing no deaths, are especially interesting. The study leaves one with a very favorable opinion regarding chemotherapy in penetrating abdominal gunshot wounds.

8. I am of the opinion that the use of the sulfonamides, especially intraperitoneally in gunshot injuries of the intestines, will lower the mortality in these victims very materially. In the present series sulfathiazole was the only sulfonamide used intraperitoneally.

9. None of the patients treated with the sulfonamides showed any evidence of toxicity attributable to these drugs.

10. Although peritoneal fluid concentrations were not determined, none of the cases being drained, the small number of blood level determinations for these drugs failed to show any high figures in spite of large doses implanted intraperitoneally in some cases.

11. It appears that the sulfonamides are especially beneficial in those cases showing "massive" hemorrhage. Such cases showed a 91.6 per

cent mortality in the group receiving no chemotherapy as compared to the 40 per cent fatality in the group receiving additional treatment with the sulfonamides.

REFERENCES

1. McIntosh, R., Wilcox, D. A., and Wright, F. H.: Results of Sulfanilamide Treatment at the Babies Hospital, New York City, J. Pediat. **11**: 167, 1937.
2. Horsley, J. S., Jr., and Chapman, D. G.: "Primary" Streptococcic Peritonitis Treated With Sulfanilamide, South. M. J. **31**: 999, 1938.
3. Long, P. H., and Bliss, E. A.: The Clinical and Experimental Use of Sulfanilamide, Sulfapyridine and Allied Compounds, New York, 1939, The Macmillan Company, p. 173.
4. Mueller, R. S.: Use of Powdered Crystalline Sulfanilamide in Surgery, J. A. M. A. **116**: 329, 1941.
5. Mueller, R. S., and Thompson, J. E.: Local Use of Sulfanilamide in Treatment of Peritoneal Infections, J. A. M. A. **118**: 189, 1942.
6. Jackson, H. C., and Coller, F. A.: Sulfanilamide in Peritoneum, J. A. M. A. **118**: 194, 1942.
7. Rippy, E. L.: The Management of Perforating Gunshot Wounds of the Abdomen, South. Surgeon **10**: 441, 1941.
8. Ravdin, I. S., Rhoads, J. E., and Lockwood, J. S.: The Use of Sulfanilamide in the Treatment of Peritonitis Associated With Appendicitis, Ann. Surg. **3**: 53, 1940.
9. Rosenburg, S., and Wall, N. M.: The Treatment of Diffuse Peritonitis by the Direct Intraperitoneal Introduction of Sulfanilamide, Surg., Gynec. & Obst. **72**: 568, 1941.
10. Wangensteen, O. H.: Aseptic Resections in the Gastro-Intestinal Tract, Surg., Gynec. & Obst. **72**: 257, 1941.
11. Moorhead, J. J.: Surgical Experience at Pearl Harbor, J. A. M. A. **118**: 712, 1942.
12. Ravdin, I. S., and Long, P. H.: The Treatment of Army Casualties in Hawaii, Army M. Bull. **61**: 1-8, 1942.
13. McGuire, C. A.: Gun Shot Wounds of the Abdomen, Southwest. J. Med. & Surg. **28**: 69, 1920.
14. Storck, A. H.: Gunshot Wounds of the Abdomen, South. Surgeon **8**: 148, 1939.
15. Wallace, C.: War Surgery of the Abdomen, Philadelphia, 1918, The Blakiston Company, pp. 21-22.
16. Loria, Frank L.: Prognostic Factors in Abdominal Gunshot Wounds, New Orleans M. & S. J. **83**: 393, 1930.
17. Oberhelman, H. A., and LeCount, E. R.: Peace Time Bullet Wounds of the Abdomen, Arch. Surg. **32**: 373, 1936.
18. Jolly, D. W.: Field Surgery in Total War, New York, 1941, Paul B. Hoeber, Inc., p. 169.
19. Loria, Frank L.: Abdomino-thoracic Gunshot Injuries, New Orleans M. &. S. J. **95**: 105, 1942.
20. Idem: The Influence of Hemorrhage in Abdominal Gunshot Injuries, Ann. Surg. **96**: 169, 1932.
21. Rippy, E. L.: Perforating Gun Shot Wounds of the Abdomen, J. A. M. A. **115**: 1760, 1940.
22. Helfrich, L. S., Cassels, W. H., and Cole, W. H.: Cortical Extract in the Treatment of Shock, Am. J. Surg. **55**: 410, 1942.
23. Ochsner, A., Gage, I. M., and Cutting, R. A.: The Value of Drugs in the Relief of Ileus, Arch. Surg. **21**: 924, 1930.
24. Wangensteen, O. H., and Paine, J. R.: Nasal Catheter Suction Siphonage; Its Use and the Technic of Its Employment, Minnesota Med. **16**: 96, 1933.
25. Bartlett, Willard, Jr.: The Concept of Pyloric Balance in Ileus Treated by Continuous Suction From the Stomach, Arch. Surg. **23**: 484, 1934.
26. Harbison, S. A., and Key, J. A.: Local Implantation of Sulfanilamide and Its Derivatives in Wounds: Its Relation to Wound Healing and to Peritoneal Adhesions, Arch. Surg. **44**: 22, 1942.
27. Sutton, Henry B.: The Intraperitoneal Use of Sulfathiazole, With Special Reference to the Production of Adhesions; Report of a Case With Secondary Intestinal Obstruction, J. A. M. A. **119**: 559, 1942.
28. Ochsner, Alton: Discussion on Intraperitoneal Chemotherapy, J. A. M. A. **118**: 199, 1942.

A CONSIDERATION OF EMPYEMA: ESPECIALLY ITS CHRONIC PHASE

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CHRONIC empyema is a disease entity which fortunately is becoming less frequent due to two reasons. First, because of the remarkable results being accomplished in the treatment of pneumonias by the sulfonamides and second, because the proper treatment of acute empyema is much better understood by the medical profession at large than it was only a few years ago.

The investigations carried out by the empyema commission of the U. S. Army during World War I did more toward placing the treatment of acute empyema in this country on a rational basis than any investigations before or since. A consideration at this time of some of the more important problems connected with the proper handling of the acute empyema is pertinent to the present discussion since, theoretically at least, chronic empyema only results from improper treatment or lack of treatment of this condition in its acute phase.

An acute empyema usually becomes chronic because of one or a combination of the following factors:

1. The empyema was inadequately drained.
2. Open drainage was resorted to too early.
3. It was drained too late.
4. Underlying pathology in the lung parenchyma feeding the pleural infection.
5. The presence of bronchial fistulas.
6. The presence of foreign bodies in the pleural cavity such as pieces of drainage material, necrotic rib, etc.
7. An apparently acute pyogenic infection of the pleura may in reality be on a tuberculous, or a neoplastic basis.
8. Occasionally a subphrenic abscess may perforate through the diaphragm and give rise to a chronic infection of the pleura.

During the early part of World War I the empyemas associated with the hemolytic streptococcal pneumonias accompanying the influenza epidemics were operated upon early with an ensuing prohibitive mortality rate. The empyema commission concluded from their studies that this was largely due to subjecting these individuals to surgery too early. Deferring operation resulted in a great improvement in mortality rate, which dropped from around 40 per cent or more to 4 per cent. Graham,[1] who was a member of this commission, has subsequently summarized the advantages to be found by waiting until the formative stage has passed and frank pus is present, by the following:

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(1) There is less danger of creating a general open pneumothorax because there is a more or less circumscribed abscess shut off by adhesions from any communication with the free pleural cavity so that during the operation the pleural cavity, properly speaking, is not entered. (2) Even if an open pneumothorax is created, its harmful effects are lessened because (a) the subsidence of the active pneumonia has the effect of making the area of the air-inlet to the lungs larger than when many of the bronchioles and much of the lung parenchyma are blocked by the pneumonic process, so that the pleural opening is incapable of producing the same amount of harm; (b) the presence of adhesions and the inflammatory thickening and induration of the mediastinum tend to make it less mobile; (c) the patient's need of oxygen is less because of a more nearly normal metabolism; (d) the respiratory compensation is more efficient since, owing to a diminished toxemia, the respiratory muscles will not become so easily fatigued. (3) The patient is in better condition to withstand whatever shock there is connected with even so slight an operation as pleural drainage. (4) There is probably less risk of creating a septicemia from absorption of organisms from the fresh operation wound, an occurrence which seemed to us during the war to have happened a few times by the finding of positive blood cultures a few hours after operation in cases in which previously there had been sterile blood cultures, the operation having been done early in the disease. As a matter of fact, a patient who has recovered from his active pneumonic process, and whose mediastinum has become thickened and stiffened by adhesions so that it no longer has normal mobility, may withstand an open pneumothorax better than a normal individual.

It is conceded by everyone that if the accumulating fluid is a source of embarrassment to the cardiorespiratory mechanism, aspiration may be done without danger and occasionally, though rarely, one may be rewarded by the gratifying result of a cure by this method. Graham further states in this respect, "If, however, one uses the aspiration method to tide the patient over the pneumonic stage, the dangerous period, to be followed by open drainage later, then he is using the procedure which one of us (E. A. G.) has been advocating since 1918."

The time to discontinue aspiration in favor of open thoracotomy is largely based on the appearance and consistency of the exudate. When it becomes definitely purulent this time has arrived. Some observers advise taking the specific gravity of the aspirated exudate and believe it is time for operative interference when this reading reaches 1.040. Empyemas from pneumococcus infection may, in general, be drained with safety earlier than those resulting from streptococcus infections because the pneumonic process is of shorter duration and the exudate in the pleura becomes more rapidly purulent.

Even though the individual survives the shock of too early an operation he is still a more likely candidate for the development of a chronic empyema than he would have been with operation at the optimum time. The collapsed lung and the displaced mediastinum leave a cavity of great size to refill and the thickening of the visceral pleura and fibrous changes that take place in the lung parenchyma may prevent sufficient lung re-expansion to accomplish this end.

While it is evident from the foregoing that early operation is to be avoided it is equally true, though an error less frequently committed, that late operation allows changes in the pleura and lung to take place that may contribute to the development of a chronic empyema. A large collection of purulent exudate compresses the lung and if left undrained for a long period of time causes thickening of the pleura and encasement of the lung that may prevent its subsequent re-expansion.

As has been stated, repeated aspiration may occasionally result in curing an empyema and so may intercostal drainage. Either method is not in keeping with the time-honored doctrine that pus calls for wide-open adequate drainage. We believe in resecting a generous piece of rib, enough to permit the introduction of a tube at least 2 cm. in diameter. As is well known, this should be done in the most dependent portion of the empyema cavity. The usual location, in an empyema that is not encapsulated, is the mid- or postaxillary line, resecting either the eighth or ninth rib. The pleural space normally extends lower but by the time the patient comes to operation the costophrenic angle has usually become obliterated by adhesions. It is important, also, not to place the opening too far anteriorly lest pocketing occur when the patient is recumbent.

Not only is it important to make an adequate primary opening into the pleura but it is equally important to maintain that opening until the pleural cavity has largely become obliterated and sterilized. This can be determined in the usual case by the amount and character of the exudate, though for the more fastidious, examination of smears and cultures of the exudate offers a more scientific if not a more practical determination of the time to discontinue drainage. An improperly placed and too small thoracotomy opening undoubtedly accounts for the majority of empyemas becoming chronic.

Foreign bodies such as a piece of drainage tube, a piece of necrotic rib, or other substances may account for an empyema not closing. Such foreign bodies may be picked up by x-ray, by digital exploration of the cavity, by a thoracoscopic examination, or by the use of a cystoscope in the absence of the appropriate instrument.

It is important to remember that the failure of an empyema to close may not be primarily due to the condition of the pleura but may be secondary to an involvement of the lung itself. Bronchopleural fistulas, when small, may be expected to close spontaneously but when large they tend to prevent re-expansion of the lung by interfering with increase in pressure in the bronchial tree during ordinary respiration, coughing, sneezing, straining, blowing, etc. Reinfection of the pleural cavity may occur by way of the fistula especially when it is associated with bronchiectasis, abscesses, or other types of infection of the lung parenchyma. Again one must not forget that the original lesion in the lung may have been a tuberculous one and the infection in the pleura is secondary to this plus a mixed infection. The same may be said for

a primary neoplastic lesion of the lung with a secondary involvement of the pleura, though the pleural fluid when present is usually tinged with blood rather than being purulent.

In considering the treatment of an acute empyema the question arises as to the relative merits of negative suction versus other methods of handling the thoracotomy tube. In 1876, Hewett[2] described a very efficient means of using a closed method of drainage and since this time there has been considerable discussion of this method and various other methods and modifications. Binnie,[3] in speaking of the closed method of empyema drainage, made this rather apt observation, "In recent years it has been frequently rediscovered and modified in unessential details."

In my own mind, at least, I am firmly convinced that negative suction applied to the pleural cavity has very definite advantages for the following reasons:

1. Maintaining a constant intrapleural negative pressure, I believe, not only prevents the lung collapsing to the degree it would with an open thoracotomy but it would appear to be of material value in assisting in re-expansion of the lung.

2. An effort is made to place the thoracotomy tube at the most dependent area of the empyema cavity but not infrequently it is found, by digital exploration after opening into the pleura, that the opening is too high. Rather than resect a rib lower I prefer to locate the most dependent spot with my finger introduced into the pleura and then insert a mushroom catheter intercostally through a stab wound into the dependent area. This catheter is subsequently used to irrigate the cavity with Dakin's solution, saline or whatever solution is in public favor at the moment. Just now the various sulfonamides seem to have captured this coveted position. Powdered sulfanilamide, 8 Gm. dissolved in 1000 c.c. of hot sterile water, or normal saline has been recommended by Adams[4] and this sounds logical but with this solution I have had no experience. With negative suction to draw the irrigation fluid out of the cavity a continuous cleansing may be carried on which no doubt aids in earlier healing.

3. There is a saving in dressings and, I believe, in length of hospital stay.

One objection to suction has been that the patient is necessarily confined to bed but this objection can be obviated if the connecting tubes are left long.

In using negative suction it is important to make the tube, as it enters the chest, airtight. This can usually be done if the incision is closed snugly about the drainage tube in layers. A silk suture is passed on either side through the tube and chest wall to hold it in place. In addition the tube is brought out through a tightly fitting sterile rubber sponge. When this is strapped to the chest without any intervening dressing there is little likelihood of a leak occurring for approximately

a week or ten days and during this time all the advantages of closed suction will, in all probability, have been accomplished.

There are various methods of securing negative suction. The one we have used most frequently has been by use of a water suction pump connected to the faucet, but an electrically driven suction pump is superior to this method. The least effective and the most troublesome one is by use of large bottles or demijohns connected in the same manner as a Wangensteen suction apparatus. It has the single advantage of practically always being available. An effort is made to maintain a negative suction of from 8 to 11 mm. of Hg. No ill effects from this amount of suction have been encountered.

Just when an acute empyema ceases to belong in this category and should be classified as chronic probably depends less upon the lapse of time than on the pathologic changes that have occurred in the structures surrounding the cavity. Inadequate drainage results in thickening of parietal and visceral pleura; too early drainage results in pneumothorax with collapse of the lung; while too late drainage brings about the same condition from compression of the lung by the accumulated fluid. Regardless of the reason for the lung being left collapsed, and in addition to changes in its surrounding pleura, fibrous changes take place in the lung itself which interfere with subsequent re-expansion. In general such changes have occurred at the end of three months and this is the period which is usually designated in dividing acute from chronic empyema.

When one is confronted with a case of empyema which begins to assume the aspects of becoming chronic the following investigations should be carried out to determine, if possible, the cause or causes for delayed healing.

1. If the patient from the onset has not been under one's care, a careful detailed inquiry into the history of the case may be extremely valuable, particularly so as related to the possibility of tuberculous infection.

2. Careful x-ray and fluoroscopic studies should be made to determine the size and location of the cavity, the condition of the parenchyma of the lung, the possibility of the presence of foreign bodies in the cavity, the mobility of the diaphragm, the thickness of the pleura, and the possibility of osteomyelitis of the ribs, etc.

Stereoscopic and Bucky x-ray pictures may add information, as well as views of the chest taken at different angles. Exposure following filling of the cavity with diodrast, lipiodol, or other solutions opaque to the x-rays is of special importance. The size and extent of the cavity and its relationship to surrounding structures can more easily be determined, as can the presence of pockets and the length and direction of the fistulous tract leading to the empyema cavity.

3. Filling the cavity with a measured amount of fluid will also assist in determining its size and if doubt exists as to the presence of a bron-

chial fistula this point can be determined by the patient coughing up some of the fluid or patient detecting the presence in his nasal passages of chlorine, if Dakin's solution is used. Warning to proceed carefully in this examination is probably unnecessary.

4. Information as to size, shape, and direction of the cavity can be obtained by probing its interior with a flexible uterine sound, a method that should not be neglected.

5. Inspection of the interior of the cavity by the thoracoscope or, if this is not available, by a cystoscope may help confirm information already accumulated by the foregoing investigations. The presence of foreign bodies may be easily detected and their removal accomplished, or a specimen of pleura may be obtained for microscopic study if this seems advisable.

6. Secretion of the cavity procured from its interior as well as sputum, if any, should be studied by smear, culture, and possibly guinea pig inoculation.

7. Bronchoscopy may reveal a stricture of the bronchus proximal to its communication with the empyema cavity, and cases have been observed where dilating the stricture has aided in curing this condition.

8. If doubt exists as to the presence of bronchiectasis, especially in relation to a bronchial fistula, bronchography may be of value in determining this point and the extent of lung resection necessary in its correction.

9. General studies of the patient should be carried out to determine his cardiac reserve, vital capacity, extent of renal damage, if any, and the necessity for blood transfusions, all to be evaluated and correlated to determine his fitness for surgery.

Again in the treatment of chronic empyema all competent observers are agreed that if adequate drainage has not been established this should be done before resorting to more radical measures. Adequate drainage plus cleansing of the cavity, preferably by Dakin's solution, plus measures directed at improvement in the patient's general condition result in cure of the empyema in a majority of instances.

When these measures have been adequately tried and success has not resulted, then obliteration of the cavity by one or a combination of several methods must be resorted to. Either the chest wall must be collapsed to meet the lung, the cavity must be filled by adjacent structures usually muscle, or the lung must be freed to enable it to re-expand to meet the chest wall. Crushing the phrenic nerve on the affected side may permit the diaphragm to rise and be of material value in diminishing the size of the empyema cavity.

When the data which have just been enumerated have been collected one can proceed in an intelligent manner to deal with presenting conditions. In general, when a thorough trial has been made with adequate drainage and cavity sterilization without success, an operation

based on the Estlander-Schede principle is the procedure that gives the most satisfactory result. We proceed something as follows: an initial incision is made over the course of the rib which has previously been removed, sufficiently large after resecting the rib and cutting away a portion of the parietal pleura to explore the interior of the cavity. One is then able to extend the subsequent incision in the proper direction from the information thus obtained. Beginning at the most dependent portion of the cavity after reflecting the skin and subcutaneous tissue the entire chest wall including ribs, parietal pleura, and intercostal vessels and muscle are removed. The object is to remove sufficient of the overlying chest wall to permit the skin and subcutaneous tissue to fall in and come in contact with the visceral pleura covering the lung. During this undecking or saucerizing process search is made for pockets surrounding the main cavity and these are also undecked. If bronchial fistulas are present they are dealt with according to size, shape, and surrounding scar tissue. Small fistulas will probably close without special attention; large ones or ones held open by much surrounding scar tissue need excision and suture of the lung or excision of also a portion of the lung if bronchiectasis or abscesses are present.

The lower portion of the empyema cavity is attacked first and adequately taken care of before proceeding upward. If the cavity is at all extensive or the patient is in poor condition the operation will have to be divided into stages. The first stage is terminated by placing several rubber tissue drains or tubes up to the apex of the remaining cavity, these being brought out through the chest wall at the most dependent portion of the cavity. After partially closing the incision large dressings are applied and held in place by elastic adhesive for its compressing effect.

One or two weeks later a second attack is made through the same incision carrying it higher anteriorly and posteriorly, as the case may be, to obtain adequate exposure. The operation may be completed at this stage or subsequently, depending upon the individual case. With the incision extending up posteriorly between the scapula and the vertebral bodies the scapula can be elevated or drawn anteriorly and cavities reaching at the apex of the pleura may be dealt with without difficulty. The success of the operation depends upon adequately saucerizing the empyema cavity so that there are no overhanging edges and no pockets left undrained, thus permitting the skin and subcutaneous tissue to come into intimate contact with the visceral pleura covering the lung. The rubber tissue drains are gradually withdrawn allowing obliteration of the cavity from above downward. Continued compression during the healing process aids in obliteration of the cavity.

The removal of a large portion of the chest wall results in some deformity but not to the extent one might imagine, except in children and young individuals whose bone growth has not been completed. We have had no experience with decortication of the lung to allow its expansion

or with muscle flaps to fill the cavity because these measures to us seem more fraught with danger and technical difficulties and possibility of failure, while the operation as described above has given satisfactory results. If there is extensive involvement of the lung parenchyma with bronchiectasis, and/or multiple lung abscesses, the involved lobe or lobes of the lung will have to be removed before permanent cure is obtained as was the case in one of the individuals here reported.

The following case reports are presented as being illustrative of some of the points brought out in the previous discussion.

CASE 1 (No. 108,900).—P. C., a male, aged 26 years, was admitted to The Queen's Hospital Aug. 30, 1937, and discharged Nov. 25, 1937. About fourteen months previous to admission to the hospital he was operated upon elsewhere for a left empyema following pneumonia. He was in the hospital at that time for about seven months and was then discharged apparently well except for occasional purulent discharge from the thoracotomy opening. He felt well for approximately six months and then developed pain in the left chest, cough, fever, and loss of weight.

On admission to The Queen's Hospital there was a small draining sinus in the midaxillary line in the region of the ninth rib on the left side. Physical examination and x-ray studies revealed dullness and cloudiness in the left lower chest which was thought to be fluid resulting from an improperly drained empyema cavity. No evidence could be elicited in the x-ray studies suggesting pulmonary tuberculosis. His temperature was from 99 to 101 to 104° F. during the next ten days. The blood count showed hemoglobin 45 per cent, red blood cells 3,000,000, leucocytes 13,400, polymorphonuclears 77 per cent, monocytes 3, small lymphocytes 16, and large lymphocytes 4. Smears and cultures of the exudate showed Gram-positive organisms (staphylococcus) and no tubercle bacilli could be found. Urine examination was negative as was the Wassermann and Kahn. The patient was given 500 c.c. of blood on September 6, and on September 7, the hemoglobin was 60 per cent and the red blood cells 3,300,000.

First Operation.—Thoracotomy: On September 10, the patient was given another 500 c.c. of blood and under local anesthesia a portion of the eighth and ninth ribs in the posterior axillary line was resected, along with a piece of pleura about 1½ inches in diameter. The parietal pleura was markedly thickened, being approximately one inch in thickness. About one pint of pus was evacuated. A large rubber tube and a No. 24 rubber catheter were inserted into the empyema cavity through the opening, the former for drainage, the latter to be used for irrigation. Irrigation of the cavity was done twice daily with Dakin's solution. No evidence was obtained of a bronchial fistula being present after irrigations. The patient's temperature soon came down to normal. On October 5, the tubes were removed, the chest was fluoroscoped, and the interior of the cavity explored by uterine sounds and inspected by thoracoscopy. The cavity was found to extend toward and behind the pericardium and upward to about the spine of the scapula. No evidence of a bronchial fistula could be seen and none was expected since there was no discomfort experienced by the patient following irrigation of the cavity. There were no foreign bodies visible. Though the cavity was seen to be multilocular, apparently there was no pocketing or walling off of cavities. Due to the greatly thickened parietal pleura and the absence of any appreciable diminution in size of the cavity following one month's drainage and dakinization it was decided that more radical operative interference was indicated. On September 28, the hemoglobin was 56 per cent, and red blood cells, 4,120,000. On October 6, he was given 550 c.c. of blood.

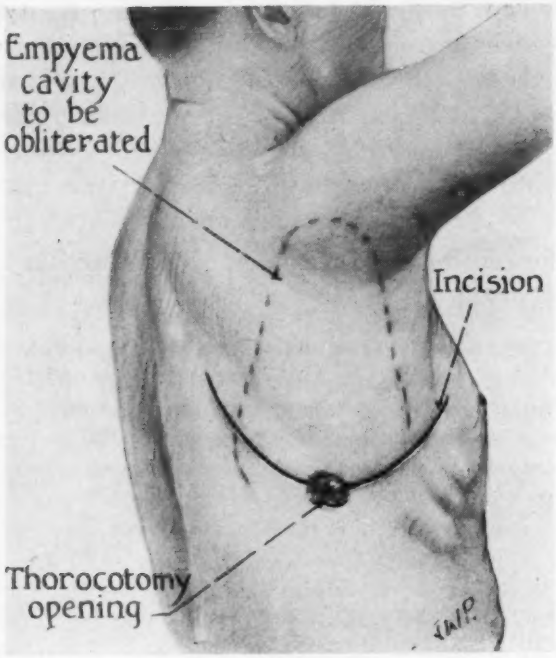
Empyema cavity to be obliterated
Incision
Thorocotomy opening

Fig. 1.

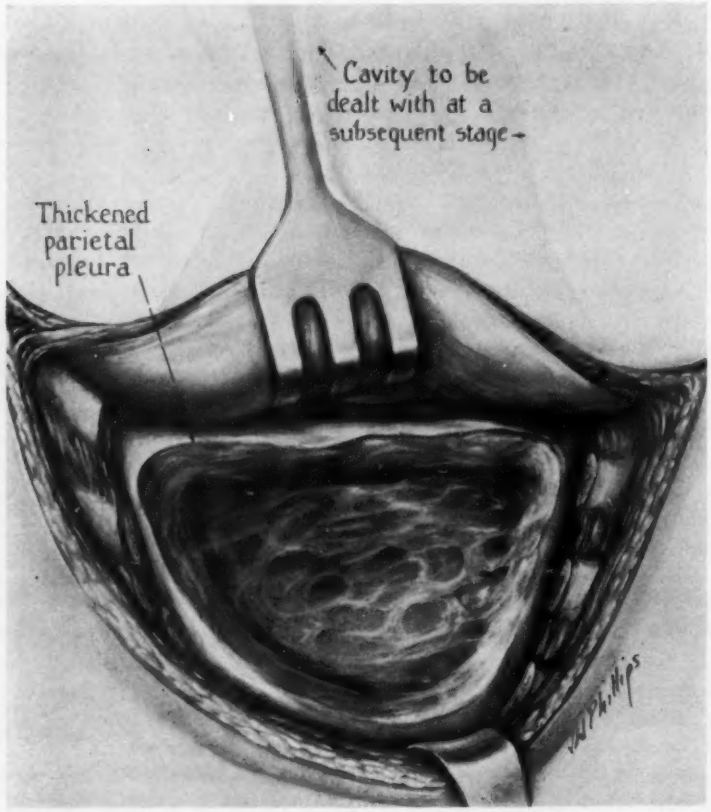
Cavity to be dealt with at a subsequent stage
Thickened parietal pleura

Fig. 2.

Second Operation.—First stage Estlander-Schede procedure: The first stage was done Oct. 8, 1937, with cyclopropane anesthesia. A curved incision was made beginning posterior to the angle of the scapula and extending down anteriorly over the ninth rib to the nipple line (Fig. 1). Portions of the eighth, ninth, and tenth ribs were resected with the underlying pleura so as to saucerize completely the lower portion of the empyema cavity (Fig. 2). The upper part of the cavity lying beneath the scapula was packed with vaseline gauze and rubber tissue drains which were brought out through the lower part of the incision and the wound was partially closed. On October 11, transfusion of 550 c.c. of blood was given. October 16, hemoglobin was 65 per cent and red blood cells 4,120,000. October 21, transfusion of 500 c.c. of blood was given.

Third Operation.—Second stage Estlander-Schede procedure. The same incision was opened and carried higher and posteriorly between the scapula and the posterior spinous processes. Portions of the fifth, sixth, and seventh ribs with underlying pleura were removed so as to complete the saucerization or undecking process over the entire empyema cavity. Drains were inserted as before and brought out through the dependent portion of the incision. The incision was partially closed. These drains were slowly removed as the operative wound healed.

Fourth Operation.—The last operation was done November 5. The phrenic nerve on the left was crushed above the clavicle. Following each removal of ribs and parietal pleura the wound was covered with large dressings and snugly strapped with elastoplast to aid in the caving-in process of the chest wall. Microscopic examination of the pleura removed at each operation showed no evidence of tuberculosis, only scar tissue and inflammatory reaction. On November 25, or 87 days following admission to the hospital, the patient was discharged with the wound practically healed. He soon returned to his former occupation, that of barber, and has been free of symptoms since.

CASE 2.—A man, aged 49 years, was admitted to Leahi Home on Dec. 21, 1937. On Nov. 2, 1937, he had been operated upon elsewhere for acute appendicitis under general anesthesia and was discharged on Nov. 13, 1937. On Nov. 23, 1937, while resting at home he had a severe attack of chills and fever with some cough, expectoration, and night sweats, and also some pain in the left chest posteriorly. He had lost during this time sixteen pounds in weight. On examination there was dullness of the entire right upper lung anteriorly and posteriorly with bronchial breath sounds but no râles, and the left lung was clear. Wassermann and Kahn were negative. Urinalysis showed a trace of albumin. Blood count was 64 per cent hemoglobin with 4,200,000 red blood cells and a white count of 10,500. Sputum on eighteen examinations was negative for tubercle bacilli. Mantoux in the first dilution was positive. X-ray showed pneumonic infiltration with cavity in the right upper lung. On Jan. 22, 1938, the patient was fluoroscoped and the right costophrenic sinus was seen to be obscured with development of early pleural effusion. This was aspirated on the following day and 50 c.c. of slightly bloody fluid were obtained, which showed on microscopic examination a 1,500 cell count, 70 per cent being polymorphonuclear cells. Culture was negative for acid-fast and other organisms. Following the pleural effusion the patient became much worse. He became extremely toxic with high septic temperature and blood count rising to 29,000 with 89 per cent polynuclear leucocytes. Another thoracentesis showed the development of a foul empyema. On Feb. 1, 1938, a portion of the tenth rib in the right posterior axillary line was removed and the empyema drained. Following this procedure and after several blood transfusions the patient's condition improved considerably. There was still expectoration of about 100 c.c. of purulent material daily and the temperature was never flat. The empyema cavity was irrigated daily but this was stopped when the patient began to expectorate fluid used for irrigating, proving the presence of bronchopleural fistula. On May 18, 1938, bronchoscopy was performed.

The findings disclosed marked hyperemia of the entire tracheobronchial tree with pus coming from the right upper lobe bronchus but no evidence of neoplasm. Because of his intermittent high elevation of temperature and failure to improve, lipiodol was instilled into the chest wall sinus. X-ray showed a sinus leading posteromedially and terminating in a large pocket. A second operation was done on June 29, 1938, at which time resection of the eighth and ninth ribs posteriorly was done and the entire empyema cavity was laid open and packed. Following this operation and another blood transfusion the patient's condition improved and his temperature dropped to normal. A right permanent phrenicectomy was performed on July 16, 1938, in an attempt to obliterate the empyema cavity and the bronchopleural fistula. Positive sputum for tuberculosis was obtained on several occasions: on Jan. 10, 1938, on guinea pig inoculation; May 18, 1938, on bronchial smear taken during bronchoscopy, and one sputum culture. Microscopic section of the removed pleura revealed one typical tubercle.

At present the empyema cavity has been completely obliterated and the external incision has healed. The final conclusion was that this individual had suffered from (1) abscess of the lung on the right which ruptured into the pleura and caused his empyema, and (2) pulmonary tuberculosis with secondary involvement of the pleura.

CASE 3.—A girl, aged 10 years, first came under my care on July 25, 1938. She had been operated upon three times, elsewhere, for drainage of a right empyema which presumably followed pneumonia. Examination revealed an obviously sick, debilitated child with a draining empyema cavity. X-ray showed a large encapsulated empyema cavity over which extensive resections had been done of portions of the fifth, sixth, seventh, and eighth ribs. Without going into the uninteresting details of the conduct of this case, which covered a period of three years and involved fifteen additional operative procedures, the points of greatest interest and of practical value will be briefly discussed.

No evidence of tuberculosis could be found in x-rays of the chest, smears and culture of the discharge, or in sections of pleura and lung subsequently excised. Examination showed bronchial fistulas brought out when the patient was asked to hold her nose and blow, and this was confirmed by irrigation with Dakin's solution and subsequently at operation. The patient's general condition was improved by transfusion, administration of vitamins, and other supportive measures.

As a first procedure more dependent drainage was instituted and while this diminished the amount of discharge, it continued. Large areas of thickened parietal pleura and ribs were resected but due to mobility of the mediastinum, the heart and other mediastinal structures moved over into the left chest and the empyema cavity persisted. The phrenic nerve was temporarily paralyzed and subsequently removed, and though this diminished the size of the cavity from below upwards it persisted and continued to pour out purulent discharge. Bronchoscopic and bronchographic examinations were done and it was concluded that bronchiectasis plus lung infection was contributing to the persistence of the empyema. The removal of the two lower lobes, or at least the portion containing the bronchial fistulas, seemed advisable. Meanwhile, due to the patient's youth, postural changes began to occur and the subsequent scoliosis resulted in the chest becoming very deformed. This has been greatly improved and prevented from developing further by braces. Attempt at removal of the lower lobes resulted in early onset of shock so that this had to be carried out in many stages. Each specimen of the lung tissue removed showed dilated bronchi with eroded and denuded epithelium and multiple nonspecific abscesses scattered about in atelectatic lung tissue.

With removal of all of the involved lung the empyema cavity has filled in and healed but the individual has been left with a much deformed thorax.

These three cases are cited in order to illustrate the usual types of chronic empyema which one may encounter. The first case was one in which the pleural cavity alone was involved, and, no doubt, had early adequate dependent and more continuous drainage been instituted, would have healed without becoming chronic. The second case probably resulted from the rupture of a lung abscess into the pleura and was complicated by pulmonary tuberculosis which, fortunately, responded to more conservative treatment than does the usual case of tuberculous empyema. The third individual represents the most serious type of nonspecific pleural infection. The empyema, no doubt, would have healed by conservative measures had it not been continuously reinfected by the underlying lung involvement.

In conclusion, I wish to emphasize the fact that there is an optimum time for operating upon infections involving the pleura and that when an adequate operation is done at this time there is little likelihood of the empyema becoming chronic.

When an empyema does become chronic the methods of procedure to determine why this has occurred are discussed and finally three representative case histories have been cited to illustrate various methods of dealing with various types of chronic empyema.

REFERENCES

1. Graham, E. A., Singer, J. J., and Ballon, H. C.: Surgical Diseases of the Chest, Chap. V. Diseases of the Pleura, Philadelphia, 1935, Lea & Febiger, pp. 113-188.
2. Hewett, F. C.: Thoracentesis; the Plan of Continuous Aspirations, Brit. M. J. **1**: 317, 1876.
3. Binnie, J. F.: The Treatment of Acute Empyema, Arch. Surg. **2**: 627, 1921.
4. Adams, F. A.: Irrigation of Wounds With Hot Sulfanilamide Solution, M. Press **205**: 318, 1941.

A NEW PROCEDURE FOR HIGH-FREQUENCY COAGULATION

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THE object of this report is to describe a method which may be used to advantage when it is necessary to produce hemostasis by coagulation, either in special or in general surgery or in experimental surgery on animals. In the usual procedure the surgeon grasps the blood vessel with a forceps, after which an assistant touches the forceps with the electrode from the high-frequency machine, the foot switch of which is operated by another assistant or nurse. With such an arrangement the surgeon lacks complete control over the manipulation, the delicacy of which must often be of a high order. Furthermore, the assistant may obscure the field of view at a time when obscurity is least desirable.

All of the above difficulties are avoided by the new procedure which entails the use of an easily detachable clip which is slipped over the upper end of the forceps that is to be used in the coagulation, the clip being connected to a wire leading from the high frequency machine. In our case the clip consists of a small piece of spring brass which was milled to take our particular forceps and bent as shown in Fig. 1. A tapered socket was made from the hilt of an old hypodermic needle and soldered into the brassclip. A tapered plug was made to fit this socket and the wire soldered to this. The wire can therefore be disconnected at any time and the forceps used in a purely surgical manner. A special clip may be designed to fit any type of forceps or clamp which it may be desired to transform into a coagulating electrode without damage to the instrument. The small radio "alligator" clip may be adapted to connect the high-frequency current to odd-shaped clamps.

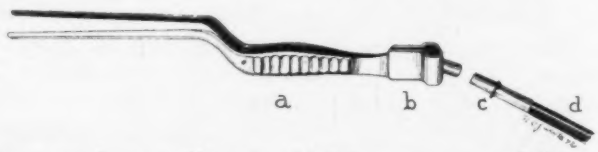

Fig. 1.—Forceps and connections for high-frequency coagulation: *a*, forceps; *b*, detachable clip; *c*, plug which is soldered to wire (*d*) and inserted into socket on clip (*b*); *d*, wire leading from high frequency machine.

In order to coagulate a blood vessel, the surgeon merely grasps the vessel with the forceps and he or another person presses the foot switch. Thus, there is avoided the crossing and interference with the operative fields by a second person, as well as the danger of moving or displacing the coagulating electrode when the contact is made in the customary way. The control of the coagulation is, therefore, directly in the hands of the surgeon, and an assistant is not necessary. Furthermore, the time spent in coagulating is reduced to one-half or less, and the loss of blood is kept at a minimum.

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Editorial

International Society of Surgery

THE International Society of Surgery, the greatest international surgical organization in the world, has transferred its headquarters from Brussels, Belgium, to the United States. This action was taken by a vote of the delegates of all of the affiliated societies of the Americas, representing Argentina, Brazil, Canada, Cuba, Ecuador, Guatemala, Mexico, Paraguay, Peru, the United States, Uruguay, and Venezuela. Headquarters of the Society will be in the Inter-America Division of the New York Academy of Medicine in New York City.

Dr. Rudolph Matas, of New Orleans, immediate past president of the International Society of Surgery and who at the present time is acting secretary and treasurer of the Society, stated in explaining the need for the change in headquarters: "The German occupation of Belgium and the Nazi devastation of the rest of Europe and all the other war-torn, ruined nations had virtually restricted the international relations of the Society to the Western Hemisphere where its fellowship is widely spread through its affiliated branches in North, Central, and South America. The executive committee of the United States division, the largest, most active contributor to its transactions, felt it a duty conjointly with their Latin American colleagues to rescue the Society out of the perils of the European conflagration. The first steps were taken in November, 1941, at Boston, but no final action could be taken to transfer the official sanctum in Brussels to America without the concurrence and approval of all the affiliated branches in America."

The executive committee which has been chosen is as follows: Dr. Elliott C. Cutler, Colonel, Medical Corps, U. S. Army, chairman in absentia; Dr. Rudolph Matas, acting secretary and treasurer; Dr. Eugene Pool; and Dr. Arthur W. Allen. At a recent meeting of the executive committee on Nov. 12, 1942, it was decided that Dr. José Arce, dean of the University of Buenos Aires, would serve as acting president of the International Society of Surgery in the absence of Professor L. Meyer, of Brussels, detained in Belgium by Nazi compulsion. At this meeting a revision of the constitution was adopted, which had been prepared previously by Dr. Matas of New Orleans.

It is of significance, particularly at this time, that the headquarters of the International Society of Surgery will be in the Inter-American division of the New York Academy of Medicine, where Dr. Enrique Cervantes, assistant secretary and treasurer of the executive committee, who is the editor of *America Clinica,* has his offices. *America Clinica* will be the official journal of the Society, so that Dr. Cervantes will be able to give particularly valuable service not only to the North American but also to the Central and South American members of the society.

—*Alton Ochsner, M.D.*
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Recent Advances in Surgery

CONDUCTED BY ALFRED BLALOCK, M.D.

AMEBIC HEPATITIS AND HEPATIC ABSCESS

AN ANALYSIS OF 181 CASES WITH REVIEW OF THE LITERATURE

ALTON OCHSNER, M.D., AND MICHAEL DEBAKEY, M.D.
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(Continued from the March issue)

Treatment.—The treatment of amebic hepatic infection is entirely surgical, although it should be realized that this may consist of either conservative or radical measures, depending upon certain developmental factors. As previously stated, the obvious rationale of this principle is based upon the fact that early recognition of an amebic hepatitis and the institution of conservative measures during this presuppurative stage may prevent progression to actual abscess formation. Moreover, even in cases in which early abscess formation has occurred, conservative measures are frequently sufficient to effect complete resolution.

The term conservative therapy is used to signify the administration of the specific drug emetine hydrochloride, with or without aspiration, depending upon the presence of indications for the latter. In early cases of amebic hepatitis, utilization of emetine alone may be sufficient (Case 1). More recently Greig[145-147] and Sarmiento[339] have directed attention to the importance of this fact. In other cases, however, a true abscess has apparently developed which requires in addition to the emetine, evacuation of its contents by aspiration (Cases 2 and 3). It is for this reason that Rogers and Megaw[326, 329] have emphasized the importance of recognizing these hepatic complications as early as possible. These facts are clearly demonstrated by Cases 1, 2, and 3 reported in this communication.

In 1911, Vedder[380, 381] undoubtedly made one of the most important advances in the therapeutic consideration of amebiasis with the introduction of emetine. Perhaps of equal significance was its subsequent popularization in amebic hepatitis by Rogers.[321-323] Of interest in this connection is the claim by Huard and associates[171] that it was used by Florence[125] thirty years earlier. Magendie and Pelletier,[229] in 1817, described emetine as an alkaloid in ipecacuanha which is the dried root of Psychotria ipecacuanha. A decoction of this root, which was originally found in Brazil but has since been cultivated in India and other

regions, was long used by the natives in the treatment of diarrhea and dysentery. Rogers and Megaw[329] state that its therapeutic use was introduced in India as early as 1660 although its popular usage did not begin until about the middle of the nineteenth century. De Savignac,[98] in 1863, demonstrated that the therapeutic efficacy of ipecac in amebiasis depended upon its alkaloid content which included principally emetine, cephalin, and psychotrine. Of these, the most powerful amebicide is emetine. The powerful amebicidal action of emetine was demonstrated, in 1912, by Vedder[382, 383] who found that amebae in vitro are killed readily by a solution of 1 to 100,000 dilution. Rogers[321-323] in the same year, reported the excellent results that may be obtained with this drug in the treatment of amebic hepatitis. Of historical interest in this connection is the fact that Maclean,[226a] as early as 1871, had directed attention to the value of ipecac in acute hepatitis and in preventing abscess formation. In 1913, James[185] demonstrated by careful experimental studies that emetine produces degenerative changes in the ameba and that the beneficial effect of the drug is due to its direct action upon the parasite. Subsequent investigators have corroborated these findings.[102, 205, 336] However, as shown by Craig,[77, 85] whereas the drug destroys the trophozoites even in great dilutions, it does not destroy the cysts except in quantities which cannot be administered safely. For this reason it is not the most effective drug in intestinal amebiasis. On the other hand, in amebic hepatitis and hepatic abscess incontrovertible evidence has demonstrated that it is the drug par excellence.

The demonstration by Rogers,[326] approximately three decades ago, that the mortality rate was decreased from 56.8 per cent to 14.4 per cent by the use of emetine and aspiration formed an important landmark in the therapeutic consideration of amebic hepatitis and hepatic abscess. Numerous observers subsequently confirmed his observations.[428] In 1917, Allen[2] reported that the admission rate for liver abscess fell from 2.4 to 1.4 per thousand troops, and the death rate from 1.3 to 0.48 per thousand within two years after adoption of ipecac by the British Army in India. Recently Spittel[353] reported a 73 per cent reduction in the incidence of liver abscess in the British Army and attributed this to the institution of early and adequate emetine therapy and to routine stool examinations. At present, it is the consensus of most authorities that emetine hydrochloride is the most valuable drug in amebic hepatic involvement and even large abscesses may completely disappear under emetine therapy alone.[429] As previously emphasized by us, every case of suspected amebic hepatitis or amebic abscess should be given the advantage of a course of emetine therapy before any other procedure is used unless rupture of the abscess appears imminent. In many cases, especially in those with early small abscesses, no other therapy will be required (Case 1). However, in the majority of cases it has been our experience that evacuation of the abscess contents will be necessary (Cases 2 and 3).

The significance of Rogers' contribution is better appreciated by a brief historical consideration of the treatment of hepatic abscess. In 1895, Matas[240] presented a succinct review of this subject and divided the history of treatment of liver abscess into three great periods: (1) "The purely medical period, during which the evacuation of the purulent collection was allowed to take place spontaneously." The mortality during this period, which embraced the history of medicine up to the sixth decade of the eighteenth century, was 80 per cent. (2) "The second epoch in the history of hepatic abscess may be said to be limited to the short period from 1860 to the early '70's, during which the early exploration with the aspirator gradually became the rule of practice, to be followed by repeated explorations and finally cautious opening with trocars or caustic pastes or by means of small incisions." (3) "The third period . . . begins with the '70's, when the Listerian discovery made the surgeons bolder in opening the peritoneal and pleural cavities in the effort to evacuate the abscess early." During this period the trocar "gradually yielded to the knife" and the "rule of surgical practice" consisted of "immediate evacuation by free incision regardless of situation or quantity of pus. . . ." That the mortality during this period was still very high is shown by Matas' report of 182 cases admitted to the Charity Hospital in New Orleans during the ten-year period, 1884 to 1894, with 87 (or 42.56 per cent) deaths. Thus, Rogers' epochal contribution may be considered as inaugurating the fourth and most important period in the treatment of amebic hepatitis and hepatic abscess.

The drug is preferably administered subcutaneously as emetine hydrochloride in daily doses of 0.065 Gm. (1 gr.) until 0.39 to 0.65 Gm. (6 to 10 gr.) have been given. Because of its toxicity in excessive dosages and its cumulative action the noxious effects of this drug should be realized and considerable care exercised in its use. Experimental investigations have shown that it is essentially a protoplasmic poison with an apparent selective action on muscle.[57, 116, 405] The danger of administering the drug in excessive dosage or over prolonged periods is indicated by the pronounced degenerative changes produced in cardiac musculature. Clinical manifestations of its toxicity consist of severe diarrhea, nausea and vomiting, profound prostration, cardiac arrhythmia and failure, muscular pains, and weakness especially in the extremities. According to Leake,[207] the maximum amount of emetine that should be given over any period of time should not exceed 10 mg. (⅙ gr.) per kg. of body weight. Accordingly, for a patient weighing 150 pounds (about 68 kg.) this would be about 0.65 mg. (10 gr.). Other amebicides such as acetarsone, carbarsone, treparsol, chiniofon (anayodin and yatren), vioform, and diodoquin which are safer and more efficient in the treatment of intestinal amebiasis are not considered desirable in the treatment of amebic hepatitis and hepatic abscess because they are not as effective in these conditions as emetine and because, as

shown by Leake,[208] they are toxic to the liver. However, immediately following completion of therapy of the amebic hepatic condition the patient should be given a course of therapy for the intestinal amebiasis. Whereas frequently this cannot be demonstrated clinically, it should be assumed to exist once the diagnosis of amebic hepatitis and hepatic abscess has been established. Moreover, the therapy employed for the latter should not be considered adequate for the treatment of the intestinal infestation. For this reason a separate and different type of therapy should be employed for the intestinal amebiasis.

Aspiration is the procedure of choice in cases in which evacuation of the abscess becomes necessary. In these cases, however, it should be realized that the preliminary administration of emetine is important. In the Tulane Surgical Service, emetine hydrochloride is administered routinely in these cases in daily doses of 0.065 Gm. (1 gr.) for two or four days preceding the aspiration. Because the majority of amebic hepatic abscesses are sterile and because of the prognostic and therapeutic importance of this fact every attempt should be made to maintain this sterility. The evacuation of these abscesses by open drainage is invariably followed by secondary infection and the morbid consequences of this development have been previously emphasized. It is possible to evacuate the abscess and simultaneously maintain its sterility only by closed drainage. In the discussion under prognosis, the contrasting mortality obtained by the two methods convincingly demonstrates the obvious advantages of closed drainage. It will be recalled that in our series of cases open drainage was used in 80 with a mortality of 22.1 per cent and conservative therapy in 83 with only three deaths, a mortality incidence of only 3.6 per cent (Graph XIII). Of interest also is the fact that aspiration was performed without preliminary emetine in two of these three fatal cases and within a relatively short period of time following the procedure the patients developed manifestations of shock and died. The other patient was treated with emetine and anayodin on the medical service for almost three weeks and no attempt made to drain the hepatic abscess. At autopsy a huge hepatic abscess measuring approximately 20 cm. in diameter was found in addition to several smaller abscesses. We are firmly convinced that these three tragedies could have been averted, the first two by the preliminary administration of emetine and the third by aspiration of the abscess in addition to the use of emetine.

As previously emphasized, the procedure of aspiration should be performed in the operating room because the facility and reliability of strict asepsis are probably greater and because open drainage can be done immediately if the abscess is found to be secondarily infected. This may be determined readily by an immediate smear of the aspirated pus which in secondarily infected cases would reveal upon microscopic examination the presence of large numbers of bacteria and leucocytes. The technique of aspiration has been described above and the procedure

is performed in a similar manner for both diagnostic and therapeutic purposes (Figs. 22, 23, 24). The administration of emetine should be continued after closed drainage of the abscess until 0.39 to 0.65 Gm. (6 to 10 gr.) have been given. As previously stated by us, it is considered unnecessary as well as undesirable to introduce any substance into the abscess cavity as the use of irrigating solutions of amebicides has been found to be valueless.[40b-d, 49, 328, 366]

Open drainage is usually necessary in secondarily infected cases as demonstrated by smear and culture. Of interest in this connection is the recent report of Alport and Ghaliougui[3] on the successful use of sulfonamides in a case of amebic hepatic abscess secondarily infected with *Bacillus pyocyaneus*. Accordingly even in the presence of secondary infection closed drainage was successfully employed. It is presumed that this was permitted by the use of sulfonamides. This is a promising development and if by this means it will be possible to employ closed drainage even in the secondarily infected cases the prognosis will be materially improved. Whereas this can be determined only by further experience, the sulfonamides should certainly be applied in all cases of secondarily infected amebic hepatic abscess. However, if open drainage becomes necessary it is imperative to institute that type of drainage which completely avoids the slightest possibility of contamination of the two virgin serous surfaces, i.e., the pleural and the peritoneal. In previous publications this phase of the subject was considered and the literature reviewed.[281, 286] In general the two types of drainage procedures which can be employed are transthoracic and transabdominal, either through a transserous or an extraserous approach. The transthoracic procedures were originally developed for the purpose of draining pyogenic abscesses of the liver or subphrenic spaces. In 1883, Trendelenburg[377] suggested this procedure and avoidance of contamination of the pleural cavity was attempted by suturing the costal and diaphragmatic layers of the pleura together. Several modifications[226, 296] of this procedure were subsequently advocated and of these probably the most popular is that of Beck[15, 16] who suggested, at the first stage operation, suturing the costophrenic pleural reflection and packing the wound for forty-eight hours with gauze impregnated with an irritative substance to produce adhesions, and at the second stage, incising through this area. Others[109, 211, 245, 246, 296, 392] have also attempted mobilization of the costophrenic angle upward, but as stated by Brown[32] this is frequently not satisfactory because of the firm adhesions resulting from the inflammatory reaction. As previously emphasized[281] such procedures are not only unnecessary, often unwarranted, frequently difficult, and deceptively dangerous, but in many instances definitely inadequate in protecting the virgin pleural cavity against invasion. The transperitoneal method of approach has similar disadvantages because it also permits contamination of uninvolved portions of the peritoneum. This is forcefully emphasized by graphic demonstrations of the contrasting

mortality statistics of the transserous and extraserous methods of drainage (Graph XV). Of 47 cases drained transpleurally, 7 had serious pleural involvement as determined by autopsy. Of the 7 deaths in the group in which patients were drained transperitoneally 6 developed generalized peritonitis and one died of spinal analgesia on the operating table. The one death in the group of patients drained extraserously has been described previously. This patient had multiple liver abscess with secondary infection and, following a second operation to drain other abscesses, developed pneumonia and died.

These facts demonstrate clearly the surgical rationale of the important principles, thoroughly appreciated over forty years ago,[12,150, 256, 346, 367] that drainage of such abscesses should be performed in such a manner that contamination of the pleural and peritoneal cavities is completely avoided. This may be accomplished best by an extraserous anterior or posterior approach, depending upon the location of the abscess. Accordingly, in those located anteriorly, the procedure described by Clairmont and Meyer[62] for drainage of subphrenic abscess should be employed. This consists briefly of making the skin incision just beneath and parallel to the costal margin, traversing the oblique muscles and transversalis fascia, and approaching the abscess extraperitoneally by carefully mobilizing the parietal peritoneum from the lower surface of the diaphragm.

In posteriorly located abscesses, on the other hand, the retroperitoneal approach previously described by us[430] is considered the most rational. This consists briefly in subperiosteally resecting the twelfth rib and entering the retroperitoneal space through a transverse incision made at the level of the spinous process of the first lumbar vertebra in the bed of the resected rib. The liver abscess is approached extraserously by carefully mobilizing the parietal peritoneum from the undersurface of the diaphragm. The comparatively low mortality of 6.6 per cent, i.e., only one death, in 15 cases of our series in which this procedure was employed convincingly demonstrated its value.

As previously stated in secondarily infected amebic hepatic abscess that is pointing, extraserous drainage may be done simply by incision over the point of greatest localization. Such a procedure in these cases becomes extraserous because the abscess has become walled off from the peritoneal cavity by involvement of the abdominal parietes. This method of drainage was possible in 19 of our cases, with two deaths, a mortality of only 10.5 per cent. The inadequate admission of emetine is believed to have been an important contributory factor in the death of these two patients. As previously emphasized, emetine therapy is just as important in cases requiring open drainage as in those in which closed drainage is used, and should be instituted in a similar manner.

It should be realized that following treatment of the amebic hepatic involvement it is essential to eliminate the intestinal infection. Failure to do this probably accounts for many recurrent abscesses. Since

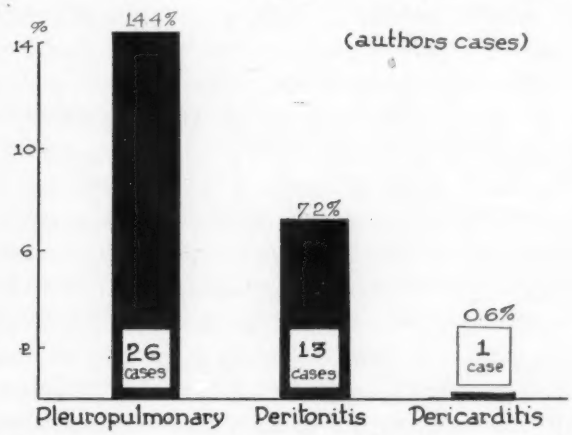
emetine is not satisfactory for this purpose, other amebicides such as anayodin or diodoquin should be employed. The comparative value of these various amebicides is beyond the scope of this presentation and have been considered elsewhere.[92, 237, 241, 266]

Complications.—The complications of amebic hepatic abscess consist essentially of secondary infection with pyogenic organisms, direct extension or rupture of the abscess into one of the adjacent viscera or serous cavities, and thrombosis and embolism. Their occurrence usually signifies negligence or procrastination on the part of the patient but occasionally reflects the physician's mismanagement. Thus, the development of peritonitis or empyema following that type of open drainage which does not completely avoid contamination of these virgin serous surfaces is obviously not due to the patient's disregard but is an example of the latter possibility. Moreover, lack of recognition of the condition sufficiently early undoubtedly accounts for the development of some of the complications. This is usually due to the absence of its consideration.

Pleuropulmonary involvement undoubtedly comprises the most frequent complication. In the majority of cases this is the result of either rupture or direct extension of the liver abscess through the diaphragm. Only a succinct discussion of the pertinent features of these complications will be given here as they have been extensively considered by us[280] in a previous publication. The incidence of pleuropulmonary involvement in amebic hepatic abscess varies considerably according to different observers. Thus, the low incidences of 3 and 3.1 per cent reported by Strong[361] and Harris,[156] respectively, are in direct contrast with the unusually high figures of 40.8 and 58.8 per cent recorded by Thompson[370] and Thierfelder,[369] respectively. In a collected series of 2,490 cases of amebic hepatic abscess pleural complications occurred in 198 (or 7.5 per cent) and pulmonary complications in 209 (or 8.3 per cent).[280] Thus, the combined incidence of pleuropulmonary complications was 15.8 per cent. In the previous series of 139 cases analyzed by us a similar incidence was obtained.[287] In the more recent series of 42 cases there were 5 cases with these complications.[285] Thus, in the total series of 181 cases, pleural complications occurred in 9 (or 5 per cent) and pulmonary in 17 (or 9.4 per cent) giving a total incidence of 14.4 per cent (Graph XVI). Of the 26 cases with pleuropulmonary involvement 8 (or 30.7 per cent) developed bronchohepatic fistula, 9 (or 34.6 per cent) empyema, and 9 (or 34.6 per cent) pulmonary abscess.

Pleuropulmonary amebiasis is occasionally due to systemic invasion but most commonly results from direct extension of an amebic hepatic abscess. Whereas it is doubtful whether primary infection of the lung without an antecedent infection of the bowel ever occurs, pulmonary metastatic lesions resulting from invasion of the systemic circulation apparently do occur rarely. Examples of the former possibility include cases of primary amebic bronchitis and pneumonitis reported by several

observers.[431] The occurrence of pleuropulmonary amebiasis resulting from systemic invasion by the ameba is convincingly demonstrated by the recent report of Girgis[139] who described two fatal cases of multiple amebic abscesses of the lung associated with multiple liver abscesses and active amebic dysentery in which the "whole series of events, . . . , including both the gross and histologic findings, may be taken as strong evidence for blood-borne amebic infections of the lungs from the liver through the right side of the heart to the lungs where we have actually found entamoebae in the emboli contained in the pulmonary arterial branches as well as in the abscess cavities." Cantlie,[40a] in 1904, had directed attention to the possible embolic origin of some amebic lung abscesses. It is the consensus of most authorities, however, that pleuropulmonary amebiasis is most commonly the result of direct extension from an amebic hepatic abscess and follows usually perforation through the diaphragm but rarely transphrenic immigration via the lymphatics.[213] The location and virulence of the amebic hepatic infection probably explain the frequency of pleural and pulmonary complications.



Graph XVI.—Relative incidences of various complications in authors' cases of amebic hepatic abscess.

The majority of amebic hepatic abscesses develop near the convex surface and in the posterior portion of the right lobe. As the abscess enlarges the adjacent diaphragm becomes incorporated in the wall of the abscess facilitating its extension into the thoracic cavity. Involvement of the pleural cavity or lung parenchyma is probably influenced by the acuteness or chronicity of the hepatic process. Accordingly, an abscess which develops slowly is likely to produce an accompanying pleurisy with adhesions which would tend to obliterate the pleural cavity and thus permit rupture into the base of the right lung (Case 4). On the other hand, if the abscess progresses rapidly the preceding pleural reaction may not be sufficiently extensive to produce obliterating adhesions and the abscess ruptures into the pleural cavity (Case 5). Not infrequently rupture into the lung parenchyma produces a bronchial fistula and the abscess contents are evacuated by coughing (Case 4).

In a previous publication we[280] presented a review of the literature and an analysis of 153 collected and 15 personal cases of pleuropulmonary amebiasis. Based upon this study pleuropulmonary amebic infections were classified into five types depending upon their gross pathologic characteristics: (1) hematogenous pulmonary abscess without liver involvement (14.3 per cent of the collected cases and none of the authors were of this type); (2) hematogenous pulmonary abscess and independent liver abscess (10.4 per cent of the collected cases and none of the authors' were of this type); (3) pulmonary abscess extending from liver abscess (these comprised 37.2 per cent of the collected and 34.6 per cent of our cases); (4) bronchohepatic fistula with little pulmonary involvement (this occurred in 19.6 per cent of the collected cases and 30.7 per cent of ours); and (5) empyema extending from a liver abscess (these formed 17.6 per cent of the collected and 34.6 per cent of our cases). It may be observed, therefore, that almost four-fifths of the cases result from direct extension of a hepatic process.

The clinical manifestations of pleuropulmonary amebiasis vary with the extent and mode of infection. The most common complaint is pain in the lower portion of the right chest with frequent radiation to the shoulder.[12, 370] This may be explained on the basis of involvement of the diaphragmatic pleura. This is especially pronounced by extension of the amebic hepatic abscess upward. The pain is sometimes described as sharp, stabbing, or shooting in character and occasionally is violent and aggravated by coughing or deep inspiration.[309] Another prominent symptom is an annoying unproductive cough which occurs particularly in the beginning. Dyspnea may be present especially when a large abscess produces considerable encroachment upon the lung field. Not infrequently the symptoms become suddenly more pronounced and the patient begins to cough up large quantities of purulent material and particularly chocolate-sauce pus which is pathognomonic of a hepatobronchial fistula. A carefully performed sputum examination will frequently reveal the ameba and establish the diagnosis. Other common manifestations are fever usually more pronounced than in uncomplicated amebic hepatic abscess, anorexia, malaise, weakness, and loss of weight. The chest findings may vary but usually are characteristic of consolidation and cavitation especially at the base of the right lung. In those cases which rupture into the pleural cavity there are characteristically present manifestations of pleural effusion. The pleurisy and pleural effusion which precede the rupture represent a contiguous inflammatory reaction.[4] There is usually a moderate leucocytosis which is more pronounced in those cases which rupture into the pleural cavity and less in the hepatobronchial fistula cases.

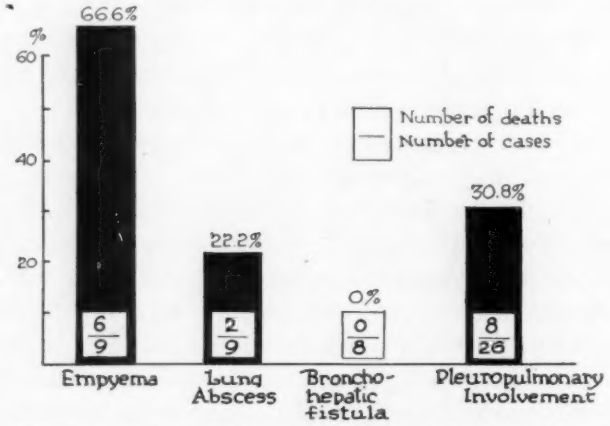
Roentgenographic examination is of particular diagnostic significance in these complications. Aside from the fluoroscopic demonstration of elevation and immobilization of the diaphragm there is characteristically a triangular shadow in both the anteroposterior and lateral roentgeno-

grams with its base toward the liver and its apex extending toward the hilum of the lung (Figs. 11 to 15). There is also an increased density in the lower lung field. In cases in which the hepatic abscess ruptures into the pleural cavity the roentgenographic picture is that of pleural effusion (Figs. 18 to 20). By the use of such contrast substances as iodized oil introduced into the trachea a bronchohepatic fistula may be readily visualized in the roentgenogram (Fig. 16).[89]

The diagnosis of these complications is usually not difficult if the condition is considered. In addition to the characteristic pulmonary manifestations, evidence of hepatic involvement is usually present. Because of the frequent chronicity and the presence of bloody sputum the condition is likely to be confused with tuberculosis. It should be realized, however, that whereas a tuberculous lesion most commonly occurs in the apex, in amebic involvement the lesion is generally at the base and associated with hepatic involvement. Moreover, sputum examination should exclude tuberculosis and the demonstration of amebae should establish the diagnosis. The diagnosis may be suspected further by the characteristic roentgenographic findings (Figs. 11 to 15, and 18 to 20). The expectoration of characteristic chocolate-sauce pus is pathognomonic (Case 4). In the cases with pleural involvement aspiration of the pleural cavity usually reveals the pus (Case 5).

The prognosis in these complications is influenced by several factors including the type of involvement and therapy employed. The prognosis is worse in those cases in which the hepatic abscess ruptures into the pleural cavity and is best in those in which a bronchohepatic fistula develops with minimal pulmonary reaction. Moreover, in the latter cases the prognosis is not as good in those in which a small communication with the bronchus and a great lung reaction exist as in those with a large communication which permits rapid evacuation.[252] In the former, type of incomplete evacuation and the presence of secondary infection increase the gravity of the condition. The prognostic influence of the type of involvement is clearly demonstrated by respective mortality rates. Thus, in the collected cases the mortality was 77.7 per cent in the liver abscess with empyema and 43.2 per cent in liver abscess with extension into the lung, whereas it was only 10 per cent in liver abscess with bronchohepatic fistula. This contrast is even more pronounced in our series of 26 cases. Whereas of 8 cases with bronchohepatic fistula none died, of 9 cases with empyema 6 (or 66.6 per cent) died and of 9 cases with lung extension 2 (or 22.2 per cent) died (Graph XVII). The total mortality in our series was 30.8 per cent which is somewhat similar to that (28.5 per cent) reported by Huard and Meyer-May[174] in their series of 21 cases. The prognostic significance of the type of therapy is demonstrated by the fact that in the collected series the mortality was 56.1 per cent in those not receiving emetine and 9.1 per cent in those treated with emetine. These respective figures in our series were 60 per cent and 0. The treatment of these pleuropulmonary com-

plications is governed by the type of involvement. Emetine should be administered in all cases just as described above in the treatment of uncomplicated hepatic lesions and its inestimable value has been emphasized previously.[233, 280] Conservative therapy consisting of emetine administration and postural drainage is usually sufficient in the bronchohepatic fistula type in which the communication is sufficient to permit rapid evacuation (Case 4). In some cases bronchoscopic drainage as suggested by Pellé[303] may be of value. The superiority of conservative therapy is shown by the fact that all of the cases in our series treated in this manner recovered, whereas only about a third of those operated upon recovered. More recently the value of conservative therapy has been emphasized by several observers.[7, 100, 147, 195, 344] However, operation may be necessary in certain cases. Thus, in those associated with an empyema aspiration should be done first and if secondary infection is demonstrated open drainage should be instituted.[40b, 40c, 40d, 203, 280] However, if secondary infection is not demonstrable, conservative therapy should be maintained (Case 5).



Graph XVII.—Relative mortality incidences in authors' cases of pleuropulmonary involvement according to type of lesion.

Extension into the peritoneal cavity is the next most frequent complication of an amebic hepatic abscess. The incidence varies according to different authors. Thus, Cyr[90] reported this eventuality in 6.9 per cent of 563 cases, Ludlow[218] in 5 per cent of 100 cases, Huard and Meyer-May[174] in 3.3 per cent of 150 cases, Jayasuriya[188] in 2.7 per cent of 73 cases, and Peiris[302] in 7.1 per cent of 28 cases. In a collected series of 1,095 cases, including ours, the hepatic abscess ruptured into the peritoneal cavity in 66 (or 6 per cent).[90, 174, 188, 218, 302] Vergoz and Hermanjat-Gerin[384] found that of 80 cases of ruptured amebic abscess, 39 (or 48.7 per cent) were into the peritoneal cavity. Of 46 cases analyzed by Acebal[1] there were 24 with generalized peritonitis and 22 with localized peritonitis. In our series of 181 cases of amebic hepatic abscess there were 13 (or 7.2 per cent) that ruptured into the peritoneal cavity and this comprised one-third of the total number of ruptured hepatic ab-

scesses (Graph XVI). It is interesting to observe that whereas in the previous series of 139 cases the incidence of this complication was 7.9 per cent, in the more recent series of 42 cases the incidence dropped to 4.7 per cent.

Depending upon the type of development, amebic hepatic abscesses that rupture into the peritoneal cavity produce localized or generalized peritonitis. Most of these develop by gradual extension of the abscess to the periphery of the inferior surface of the liver stimulating a peritoneal reaction and permitting the formation of peritoneal adhesions and consequent localization. On the other hand, if the abscess extends and enlarges more rapidly, especially if secondary infection develops, and particularly if there is a precipitating factor such as trauma or exertion, perforation and rupture of the abscess may occur into a virgin peritoneal cavity producing a generalized peritonitis. Accordingly, the clinical manifestations will vary. Whereas in the former type in which a previous peritoneal reaction has permitted walling off of the abscess resulting in relatively mild manifestations, in the latter in which rupture occurs into a virgin peritoneal cavity with good absorbability, severe clinical manifestations are produced by the rapid absorption of the toxic material. The danger of sudden rupture of an abscess into a virgin peritoneal cavity has been emphasized previously.[276] Because of this rapid absorption of the toxic material, the clinical manifestations in these cases are characteristically those of shock. Following the experience of sudden intense abdominal pain the patient appears anxious, the face flushed, the pulse rapid and feeble, the blood pressure drops, the temperature begins to rise percipitously, generalized abdominal tenderness and rigidity become manifest, and there is all the evidence of an acute abdominal catastrophe. Whereas the manifestations may not be as severe as in primary pyogenic infections, nevertheless the prognosis is relatively bad. Thus, in a collected series of 46 cases there were 28 (or 60.8 per cent) deaths.[1] Loubejac[215] reported 11 cases with 3 (or 27.3 per cent) deaths. In our series of 13 cases there were 10 (or 77 per cent) deaths. In the case with localized peritonitis the prognosis is better.

The presence of a localized or generalized peritonitis also influences the therapy, although emetine should be administered in all cases. In the former aspiration should be done first and if secondary infection is demonstrated, immediate open drainage should be instituted. This is necessary in the majority of cases and was done in the three cases of our series. In the remaining cases the patients died soon after the onset of generalized peritonitis. Biggam and Rajol[23] advocate conservative therapy consisting of aspiration of the peritoneal fluid and the administration of emetine. On the other hand, Loubejac[215] prefers drainage by operation.

Occasionally an amebic hepatic abscess may rupture into the pericardial cavity. This unusual complication occurs in less than 2 per cent

of the cases. In Huard and Meyer-May's[174] series of 150 cases it was present in 3 (or 2 per cent) and in our series of 181 cases it occurred in only 1 (or 0.6 per cent) (Graph XVI). Of 80 ruptured amebic hepatic abscesses reported by Vergoz and Hermenjat-Gerin,[384] 13 (or 16.2 per cent) perforated into the pericardium. They are more likely to follow abscesses of the left lobe of the liver, which are more difficult to recognize. Clinically this complication is characterized by sudden manifestations of pyopericardium with a rapidly fatal termination. All of Vergoz and Hermenjat-Gerin's[384] cases were fatal as were ours. However, a few recoveries have been reported. Huard and Meyer-May[173] recorded a patient who recovered following open drainage and more recently Purcell[310] reported a case in which conservative therapy consisting of emetine and aspiration was apparently successful.

There are still other rarer complications of amebic hepatic abscess, most of which consist of perforation. Accordingly, cases have been reported in which the abscess ruptured into the gastrointestinal tract,[51, 66, 188, 223, 261] into the suspensory ligament,[307] through the abdominal wall,[59, 174, 218, 302] into the biliary tract,[29, 69, 96] and into the portal vein and inferior vena cava.[26, 56, 64, 120, 132, 224] Perforation or drainage of an amebic hepatic abscess through the abdominal wall is one of the most frequent causes of cutaneous amebiasis and comprises from one-third to one-half of the cases of this type of amebiasis.[111, 179, 236, 267, 376, 400] We have previously reviewed this subject.[282-284] The condition is characterized by progressive necrosis and ulceration of the skin. The treatment consists essentially of radical excision and administration of emetine.

Other even rarer complications are cerebral abscess and extension of an amebic hepatic abscess into the spleen and the kidneys. Kartulis[192] found brain abscesses in 3 per cent of the cases of amebic dysentery in Egypt, and Izar[184] collected 56 cases of amebic abscesses of the brain, 29 of which occurred in Egyptians. Cerebral abscess is considered the result of blood-borne infection and hepatic and pleuropulmonary involvement are frequent antecedent lesions.[311] Runyan and Herrick[335] reported a case of amebic cerebral abscess which occurred shortly after drainage of a liver abscess and sponging of the cavity with gauze. The condition is almost invariably fatal. Of the 59 cases collected by Huard[170] all terminated fatally. The rarity of splenic involvement is shown by the fact that Huard[170] was able to collect only 6 cases from the literature. It may be due to metastasis from the intestine or to direct extension of an amebic abscess of the left lobe of the liver. Kidney lesions are also extremely rare. These complications are usually determined at autopsy and are associated with the more severe extensive and neglected cases of amebiasis.

As previously stated, it is significant that most of these complications occurred in the earlier reported cases. At the present time because of

the rational conception and specific therapy of amebiasis these complications should seldom if ever occur.

ILLUSTRATIVE CASE REPORTS

The following case is a characteristic example of an amebic hepatitis with satisfactory response to conservative therapy. This is considered to be in the "presuppurative" stage and by the use of emetine progression to abscess formation is prevented. Upon admission the diagnosis was not clear and certain features indicated the possibility of bronchopneumonia and pleurisy. For this reason he was placed on sulfapyridine therapy. It soon became apparent that this diagnosis was incorrect and the presence of amebae in the stool suggested the correct diagnosis which was confirmed by the dramatic response to emetine therapy.

CASE 1.—R. J., a colored male, 33 years of age, was admitted to Charity Hospital on Feb. 3, 1940, and discharged on Feb. 24, 1940.

Chief Complaint.—Sharp pain in right lower chest and upper abdomen.

Present Illness.—The patient stated that his illness began on Jan. 30, 1940, at which time he had a pain high in the epigastrium. This was a drawing type of pain associated with a fullness in the abdomen. The pain developed gradually and was soon accompanied by slight nausea. The patient had to gag himself with his fingers to vomit, but the nausea was not relieved by vomiting. The pain in the epigastrium and the nausea persisted for several days and then he developed chills and fever. The next day the patient went to work as usual in a lumber yard, but felt very tired and his back ached severely. He claims that his joints ached so much that he could not bend his knees. The following night a severe, sharp pain struck him in the right hypochondriac region. The pain was so severe that it caused him to double up. Deep breathing aggravated the pain which sometimes radiated to the right shoulder. He remained in bed until he came to the hospital. After admittance he had a bad cold and a running nose for about a week. He coughed occasionally. The cough was slightly productive of white watery sputum. His appetite has been good until the writing of this report. He had a history of chronic constipation but no diarrhea except occasionally a loose stool.

Physical Examination.—Pulse, 100; respiration, 24; blood pressure, 140/80; temperature, 102° F. The patient was apparently not acutely ill; he lay quietly in bed on a back rest, but with a tired, worried facies. Examination of the chest revealed no significant findings except a slight lag in the right lower portion, and extreme tenderness in this area as well as decreased resonance, decreased breath sounds, and occasional dry râles. The abdomen appeared flat, muscular, and somewhat rigid. Extreme tenderness was elicited just below the costal margin on the right side and over the liver which was palpable two fingerbreadths below the right costal margin.

Laboratory Findings.—On Feb. 3, 1940, hemoglobin was 75 per cent; red blood cells, 4,100,000; white blood cells, 24,000; polymorphonuclears, 84 per cent; lymphocytes, 14 per cent. On April 7, 1940, red blood cells were 3,333,000; white blood cells, 17,400; polymorphonuclears, 84 per cent; lymphocytes, 10 per cent; monocytes, 5 per cent; urinalysis, no abnormal findings. Blood chemistry was normal, and on Feb. 8, 1940, sulfapyridine level, 12; blood Wassermann, negative; sputum, negative for all types of pneumococci and for acid-fast organisms. Stool examination on Feb. 15, 1940, showed cysts of *E. histolytica* and blood culture, negative. Roentgenographic studies of the chest on Feb. 7, 1940, showed slight thickening of the pleura in the right costophrenic angle and of the right intralobar pleura but no evidence of pneumonia or encapsulated fluid. The impression was thickened pleura.

On Feb. 16, 1940, views of the chest show clear lung fields, but there is elevation of the right leaf of the diaphragm and apparent thickening of the pleura in the right axillary region with slight obliteration of the cardiophrenic angle in the posteroanterior view (Fig. 2), and a distinct bulging of the diaphragm and obliteration of the anterior costophrenic angle in the lateral view (Fig. 3).

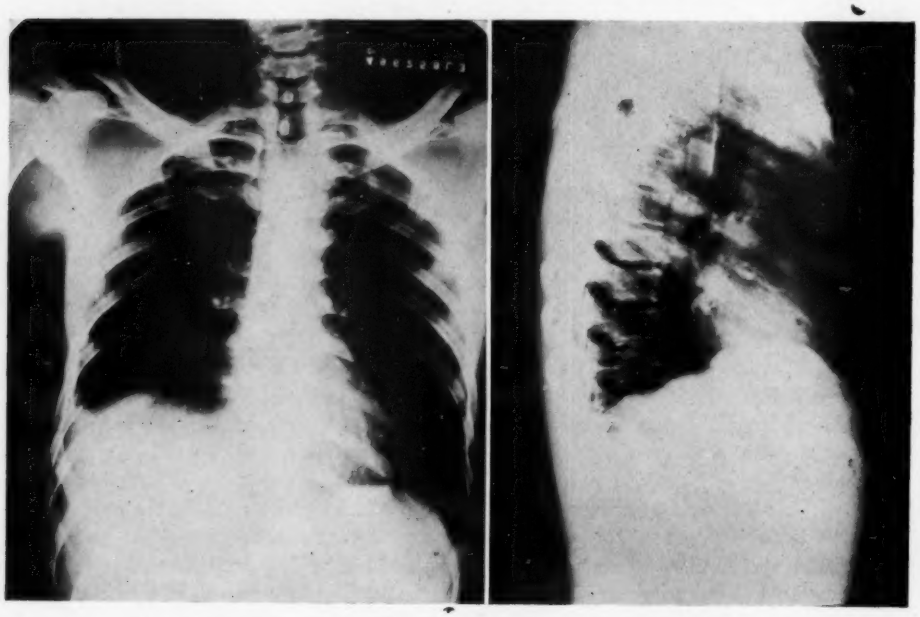


Fig. 2. Fig. 3.

Fig. 2.—Anteroposterior roentgenogram of chest of patient reported in Case 1 with amebic hepatitis showing characteristic elevation of right leaf of diaphragm with obliteration of cardiophrenic angle.

Fig. 3.—Lateral roentgenogram of chest of patient referred to in Fig. 2 showing characteristic elevation and slight bulging of right leaf of diaphragm with obliteration of anterior costophrenic angle.

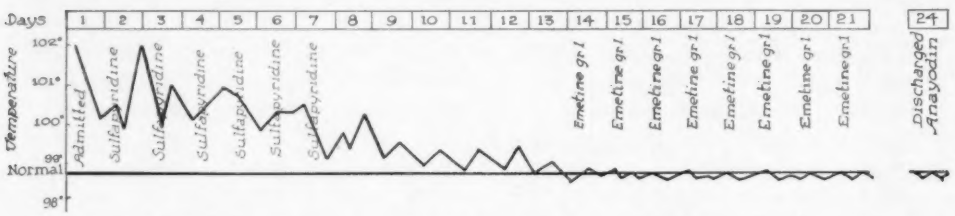


Fig. 4.—Temperature chart of patient in Case 1 showing little response to sulfapyridine but dramatic response to emetine.

Clinical Course.—The patient was admitted to the ward with the diagnosis of pleurisy with effusion and pneumonitis. He was given sulfapyridine for approximately one week but the clinical manifestations were not remarkably relieved although the temperature curve showed a slight decrease (Fig. 4). Following the demonstration of *E. histolytica* cysts in the stools and characteristic roentgenographic findings on Feb. 16, 1940, a tentative diagnosis of amebic hepatitis was made and the patient was given emetine hydrochloride, gr. 1, daily for one week. Immediately after instituting this therapy the temperature dropped to normal and remained there until his discharge (Fig. 4). The clinical manifestations were completely relieved and upon his discharge he was placed on anayodin therapy for intestinal amebiasis.

The following case is a characteristic example of an amebic hepatic abscess that responded satisfactorily to conservative therapy consisting of emetine and aspiration of the abscess. In this case the clinical manifestations were quite characteristic, including the history of an antecedent diarrhea although stool examinations were negative. The clinical diagnosis of amebic hepatic abscess was fairly evident and supported by the characteristic roentgenographic findings. The diagnosis was definitely established by the aspiration of typical chocolate-sauce pus as well as the patient's dramatic response to this conservative therapy.

CASE 2.—C. L., a white male, 56 years of age, was admitted to Charity Hospital on Jan. 2, 1941, and discharged on Jan. 28, 1941.

Chief Complaint.—Pain in the upper right abdomen for one and one-half months.

Fig. 5.

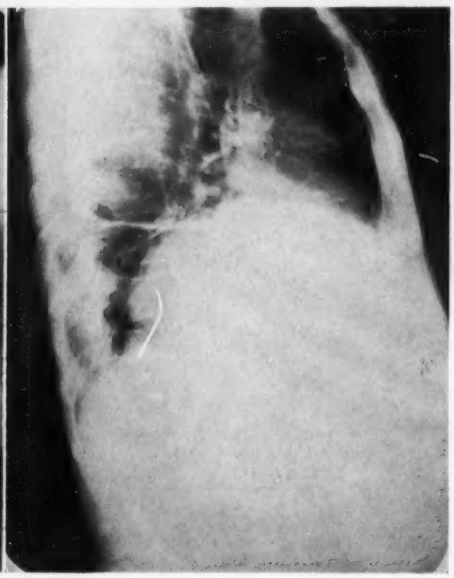


Fig. 6.

Fig. 5.—Anteroposterior roentgenogram of chest of patient reported in Case 2 with amebic hepatic abscess showing characteristic elevation of right leaf of diaphragm with distinct bulging of the diaphragm pointing into the lower lung field and obliteration of cardiophrenic angle.

Fig. 6.—Lateral roentgenogram of chest of patient referred to in Fig. 5 showing pronounced elevation and bulging upward of right leaf of diaphragm and obliteration of anterior costophrenic angle.

Present Illness.—Patient states that about three months previously he had an attack of diarrhea with twenty to thirty bowel movements daily, associated with tenesmus and mucus in the stools. Upon seeing his physician he was told to change his diet and was given medicine to relieve the diarrhea. This lasted for approximately two months and during the past month he has had fever, and pain and tenderness in the right upper abdominal quadrant. He also complains of anorexia, loss in strength, and considerable loss in weight.

Physical Examination.—Blood pressure, 120/75; temperature, 101° F.; pulse, 130; and respiration, 28. The general appearance was that of a poorly nourished man, resting quietly in bed, and not apparently acutely ill. Significant findings were limited to the lower right chest and abdomen. There semed to be a slight

lagging of the right chest and liver dullness extending higher than normal. There was marked tenderness along the right costal margin, and some rigidity over the hepatic area. The liver was enlarged two fingerbreadths below the right costal margin.

Laboratory Findings.—On Jan. 5, 1941, hemoglobin was 65 per cent; red blood cells, 3,550,000; white blood cells, 22,300; polymorphonuclears, 90 per cent; lymphocytes, 8 per cent; monocytes, 2 per cent. On Jan. 7, 1941, white blood cells were 16,500; polymorphonuclears, 85 per cent; lymphocytes, 14 per cent; monocytes, 15 per cent; and urinalysis, negative. Blood chemistry was normal and blood Wassermann negative. On Jan. 31, 1941, pus from liver abscess showed no parasites and no bacterial organisms. Stool examinations on three occasions, January 4, 6, and 13, showed no ova or parasites. Roentgenographic examination of the chest, Jan. 2, 1941, showed elevation of the right leaf of the diaphragm and atelectasis at the right base. Subsequent views of the chest showed immobilization, elevation, and localized bulging of the right leaf of the diaphragm with obliteration of the cardiophrenic angle in the posteroanterior view (Fig. 5), and obliteration of the anterior costophrenic angle in the lateral view (Fig. 6).

Clinical Course.—Following the characteristic roentgenographic findings a tentative diagnosis of amebic hepatic abscess was made and the patient started on emetine therapy. Two days later, under local analgesia, aspiration of the liver was performed and 700 c.c. of chocolate-sauce pus were removed from the liver abscess. The day after the aspiration the patient's temperature which had been ranging between 99° and 101° F. dropped to normal and remained there (Fig. 7) until his discharge from the hospital and he was completely relieved of all previous manifestations. Upon his discharge he was placed on diodoquin therapy for intestinal amebiasis.

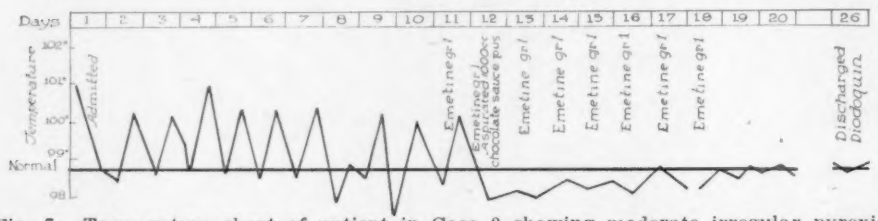


Fig. 7.—Temperature chart of patient in Case 2 showing moderate irregular pyrexia and dramatic response to emetine and aspiration therapy.

The following case is an example of amebic hepatic abscess which was at first believed to be an amebic hepatitis. The clinical manifestations of an amebic hepatitis were quite characteristic, including an antecedent dysentery and the presence of ameba in the stools. The roentgenographic findings were also characteristic. Accordingly a diagnosis of amebic hepatitis was made and the patient placed on emetine therapy. Her immediate response was dramatic; the temperature dropped to normal and she was remarkably improved. However, within about one week after beginning this therapy there was a slight recurrence of fever which was increasing and the hepatic tenderness although diminished was still present. It was soon evident that the pathologic process had progressed beyond a simple hepatitis to true abscess formation. This was definitely established by the aspiration of typical chocolate-sauce pus and the subsequent complete relief of all manifestations.

CASE 3.—M. J., a colored female, 34 years of age, was admitted to Charity Hospital on May 2, 1942, and discharged on June 14, 1942.

Chief Complaint.—The chief complaint of the patient was pain in the right upper abdomen.

Present Illness.—About two weeks prior to admission the patient began having pain in the right upper abdomen. It was dull aching in character, gradually becoming worse, and particularly aggravated by deep breathing. One week later she began having nausea and vomiting and some fever. During the six weeks prior to admission she had lost about eighteen pounds in weight. Her appetite had steadily diminished and she had lost strength. About a year ago she had "loose bowels" and passed blood in her stools. This attack lasted about a week and since then she has had no bowel disturbances.

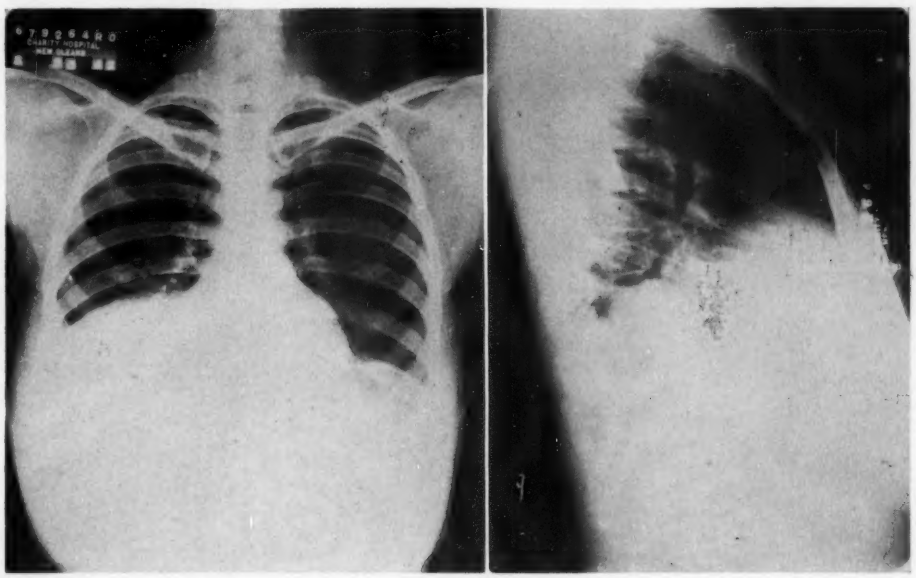


Fig. 8. Fig. 9.

Fig. 8.—Anteroposterior roentgenogram of chest of patient reported in Case 3, showing pronounced elevation of right leaf of diaphragm and obliteration of cardiophrenic angle.

Fig. 9.—Lateral roentgenogram of chest of patient referred to in Fig. 8, showing elevation of right leaf of diaphragm and obliteration of anterior costophrenic angle.

Physical Examination.—Temperature was 100.6° F.; pulse, 100; respiration, 24; blood pressure, 110/76. General observations: the patient was slightly undernourished, ambulatory, and not acutely ill. The significant findings were limited to the lower right chest and abdomen. There was some lag of the right chest and the liver dullness extended up to the fourth interspace. There was distinct muscular rigidity in the right upper abdominal quadrant and acute tenderness over this area, especially along the right anterior costal margin. The liver was palpable about three fingerbreadths below the right anterior costal margin.

Laboratory Findings.—On May 5, 1942, hemoglobin was 70 per cent; red blood cells, 3,400,000; white blood cells, 14,760; polymorphonuclears, 76 per cent; lymphocytes, 15 per cent; monocytes, 7 per cent; eosinophiles, 2 per cent; and urinalysis showed a slight trace of albumin. The blood chemistry was normal, Wassermann, negative, and stool examination revealed cysts of *E. histolytica.* Roentgenographic studies showed distinct immobilization and elevation of the right leaf of the diaphragm with obliteration of the cardiophrenic angle in the posteroanterior view (Fig. 8) and the anterior costophrenic angle in the lateral view (Fig. 9).

Clinical Course.—A diagnosis of amebic hepatitis was made and the patient placed on emetine therapy, 1 gr. daily for 10 days. Within four days the temperature dropped to normal and remained there for about one week and then there was slight recurrence of fever (Fig. 10). Although there was considerable improvement clinically the patient still complained of some pain and tenderness over

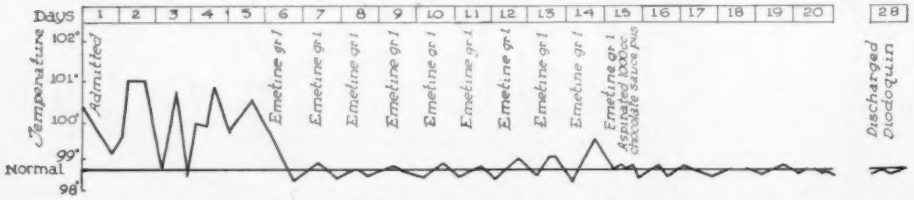


Fig. 10.—Temperature chart of patient in Case 3 showing definite response to emetine therapy with slight recurrence of fever which completely disappeared following aspiration of 1,000 c.c. of chocolate-sauce pus.

Fig. 11.

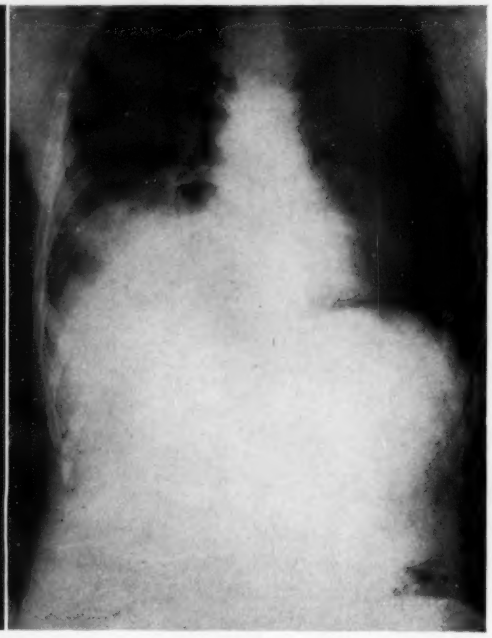


Fig. 12.

Fig. 11.—Lateral roentgenogram of chest of patient with amebic hepatic abscess made several days before rupture into lung. The obliteration of the anterior costophrenic angle and the localized upward bulging of the diaphragm are characteristic features.

Fig. 12.—Anteroposterior roentgenogram of chest of patient referred to in Fig. 11 taken several days after amebic hepatic abscess had ruptured into lung and patient began coughing up chocolate-sauce pus. There is a pronounced elevation of the right leaf of the diaphragm and obliteration of the cardiophrenic angle with the apex of a triangular shadow extending toward the hilum of the lung. The edges of the shadow are less sharply defined than in that taken before rupture, probably due to the associated pneumonitis.

the hepatic area. It was decided that the patient had a true abscess and this was subsequently verified by the aspiration of 1000 c.c. of chocolate-sauce pus. Immediately following this procedure the temperature dropped to normal and remained there until the patient was discharged when she was placed on diodoquin therapy (Fig. 10). All previous manifestations were completely relieved.

The following case is a characteristic example of an amebic hepatic abscess which ruptured into the bronchus. This patient had been ad-

mitted to the hospital on several occasions and the diagnosis was not suspected until after he began coughing up characteristic chocolate-sauce pus. The diagnosis of bronchohepatic fistula was definitely established by roentgenograms taken after intratracheal instillation of lipiodol. The satisfactory response to conservative therapy and postural drainage is clearly demonstrated.

CASE 4.—L. A., a colored male, 45 years of age, was admitted to the hospital on April 11, 1941.

Chief Complaint.—There was pain in the right chest, cough and expectoration of bloody mucus.

Fig. 13.

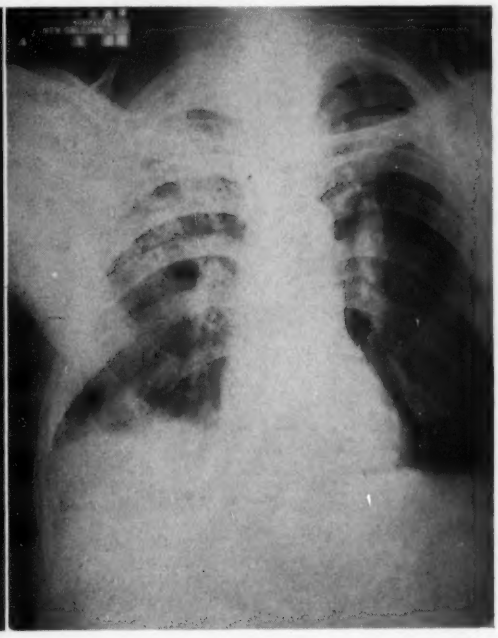


Fig. 14.

Fig. 13.—Lateral roentgenogram of chest of patient referred to in Fig. 12 and taken at same time, i.e., several days after amebic hepatic abscess had ruptured into lung. In contrast with that taken before rupture as shown in Fig. 11, it will be observed that the apex of the localized bulging is now less distinct and extends up to the hilum of the lung.

Fig. 14.—Anteroposterior roentgenogram of chest of patient reported in Case 4 with amebic hepatic abscess and bronchohepatic fistula. There is definite elevation of the right leaf of the diaphragm, obliteration of the cardiophrenic angle, characteristic triangular shadow with the apex pointing into the lower lung field, and evidence of right pneumonitis.

Present Illness.—The patient stated that he first became ill in July, 1939, with malaise, night sweats, weakness, and expectoration of bloodstained mucus. He also had pain in the right chest. He came to Charity Hospital in August, 1939, and remained there for about one month. He left the hospital feeling much better but was readmitted on several occasions since then with the same complaints. He has continued to cough and expectorate a dark reddish mucopurulent material which is sometimes tinged with bright blood. He lost about thirty-five pounds in weight since the onset of his illness. At the time of the writing of this communication, his appetite was good and he had no digestive disturbances.

Physical Examination.—Temperature was 102° F.; pulse, 90; respiration, 22, and blood pressure, 105/60. General observation revealed a somewhat undernourished

negro male apparently chronically ill. Significant findings were limited to the chest and abdomen. There was slight lagging of the right chest. The breath sounds on the right side were diminished in the lower part with dullness and moist râles in this area. Examination of the abdomen revealed tenderness immediately below the right costal margin. The liver was not palpable.

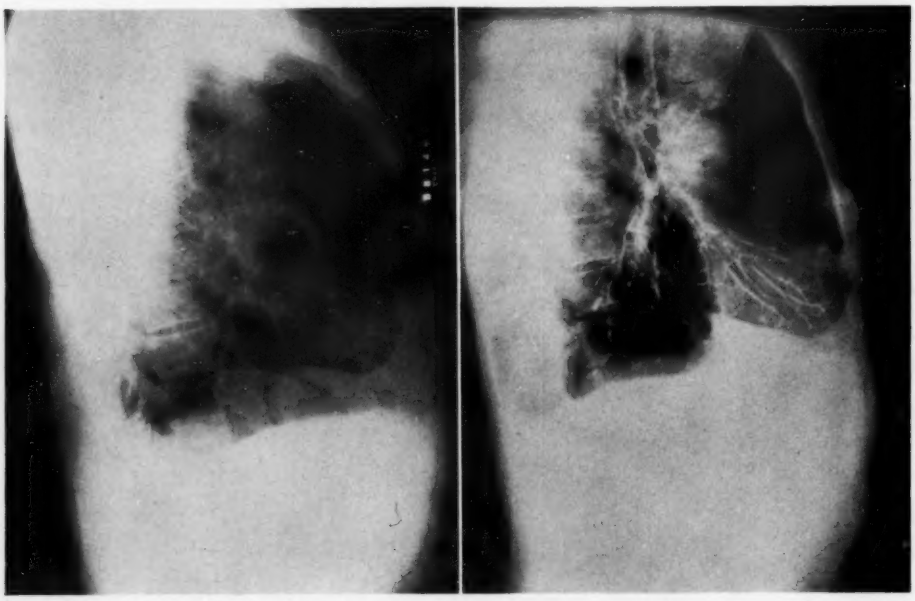


Fig. 15. Fig. 16.

Fig. 15.—Lateral roentgenogram of chest of patient referred to in Fig. 11 showing definite elevation of right leaf of diaphragm, obliteration of anterior costophrenic angle, and a characteristic triangular shadow with its apex projecting toward the hilum of the lung.

Fig. 16.—Lateral roentgenogram of intratracheal lipiodol instillation of chest of patient referred to in Figs. 11 and 12 showing bronchohepatic fistula. The lipiodol can be traced through the apex of a triangular shadow and beneath the diaphragm into the liver abscess.

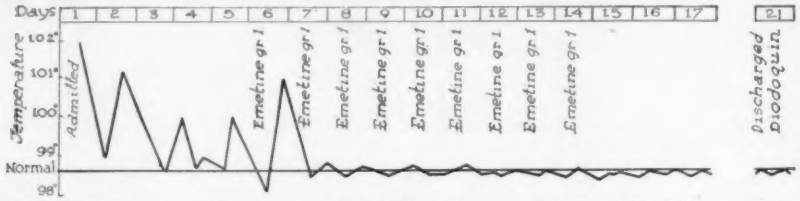


Fig. 17.—Temperature chart of patient in Case 4 showing definitive response to postural drainage and emetine therapy.

Laboratory Findings.—Urinalysis was negative, Wassermann, negative and blood culture, negative. Sputum showed no parasites and no acid-fast organisms. Hemoglobin was 60 per cent; red blood cells, 2,775,000; white blood cells, 6,750; polymorphonuclears, 76 per cent; lymphocytes, 20 per cent; monocytes, 4 per cent, and stool, negative. In the roentgenographic examination the views of the chest showed thickened pleura in the right axillary region and thickening of the interlobar pleura. The right leaf of the diaphragm was elevated and immobile with obliteration of the cardiophrenic angle in the posteroanterior view of the apex of a triangular bulging of the right diaphragm pointing toward the hilum of the lung (Figs. 14 and 15). Intratracheal lipiodol instillation revealed bronchohepatic fistula (Fig. 16).

Clinical Course.—On the basis of the characteristic clinical manifestations and the roentgenographic findings a diagnosis was made of amebic abscess of the liver with rupture into the lung and development of hepatobronchial fistula. The patient coughed up characteristic chocolate-sauce material. Therapy consisted of postural drainage and the administration of emetine hydrochloride, gr. 1, daily for nine days. He responded satisfactorily with the temperature (Fig. 17) dropping to normal and remaining there until his discharge and complete relief of manifestations.

The following case is an example of an amebic hepatic abscess that ruptured into the right pleural cavity. The clinical manifestations and the roentgenographic findings at first suggested the diagnosis of bronchopneumonia with pleural effusion and the patient was treated with sulfathiazole. His response was not satisfactory and the correct diagnosis

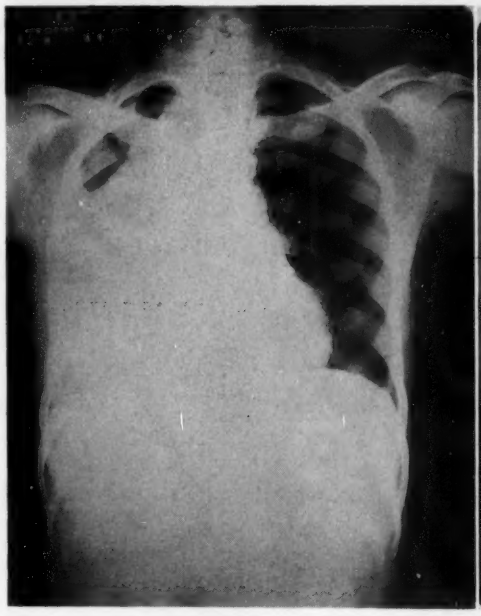


Fig. 18.

Fig. 19.

Fig. 18.—Anteroposterior roentgenogram of the chest of the patient reported in Case 5 showing opacity of right hemithorax in the lower two-thirds due to rupture of amebic hepatic abscess into pleural cavity.

Fig. 19.—Lateral roentgenogram of the chest of the patient referred to in Fig. 18 and taken at the same time. The right leaf of the diaphragm cannot be delineated.

was suggested by the finding of ameba in the stools and the demonstration by thoracentesis of chocolate-sauce pus in the right pleural cavity. Since smear and culture studies revealed the pus, sterile conservative therapy consisting of emetine and aspiration evacuation was instituted. The satisfactory response to this therapy is clearly demonstrated.

CASE 5.—P. L., a colored male, 30 years of age, was admitted to Charity Hospital on Aug. 25, 1941.

Chief Complaint.—Pain in the right chest was the chief complaint of the patient.

Present Illness.—The patient stated that about two weeks prior to admission he experienced a sudden sharp pain in the right upper abdomen and lower chest while

lifting a crate of oranges. This forced him to quit working. He used turpentine as a "rub medicine" to the painful area. However, he obtained no relief and the pain began to extend higher up into his chest. It was especially aggravated on deep inspiration and he gradually developed slight dyspnea. He stated that one week before he experienced this sudden pain he had a "cold" in his right chest. He did not think he had much fever or chills. He had a cough for several weeks and began expectorating some mucopurulent material, sometimes brownish tinged. His appetite had not decreased very much but he lost about twenty-five pounds in one month. He was slightly constipated and had never had diarrhea for a prolonged period.

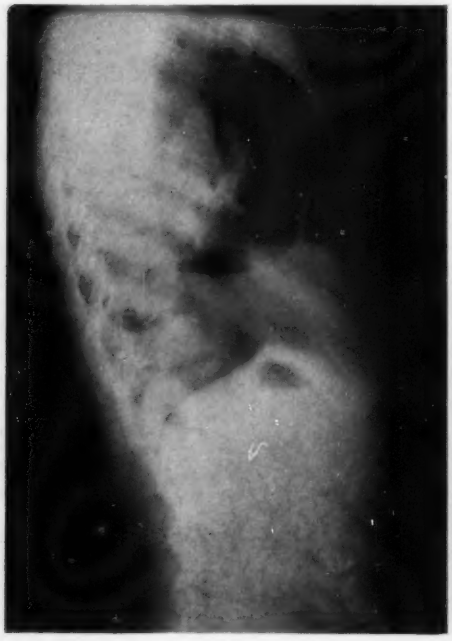


Fig. 20.

Fig. 21.

Fig. 20.—Lateral roentgenogram of the chest of the patient referred to in Figs. 18 and 19 taken after diagnostic thoracentesis which revealed chocolate-sauce pus. The fluid level may be clearly observed in the right pleural cavity.

Fig. 21.—Lateral view of the chest of the patient referred to in Figs. 18, 19, and 20 taken about one month after emetine and aspiration therapy. The right leaf of the diaphragm which is still somewhat elevated can now be delineated and the opacity in the chest has practically cleared. The patient was symptomless at this time and considered sufficiently recovered to be discharged.

Physical Examination.—Temperature was 99.4° F.; pulse, 120; respiration, 40; blood pressure, 100/70. The patient was a well-developed but somewhat poorly nourished colored male, lying quietly in bed apparently not acutely ill but having a slightly worried facies. Significant findings were limited to the chest and abdomen. There was a distinct lagging of the right side of the chest with increased tactile fremitus and dullness below the right second rib laterally and posteriorly. Resonance was definitely impaired on this side with almost absent breath sounds and a few fine râles. The heart seemed to be shifted to the left. Definite tenderness and rigidity were present in the right upper abdominal quadrant and the liver was palpable three fingerbreadths below the costal margin.

Laboratory Findings.—Hemoglobin was 65 per cent; red blood cells, 3,470,000; white blood cells, 17,000; polymorphonuclears, 73 per cent; lymphocytes, 18 per cent; monocytes, 9 per cent; blood chemistry, normal; urinalysis, negative; and Wassermann, negative. Bacteriologic examination of aspirated pleural fluid showed it to be

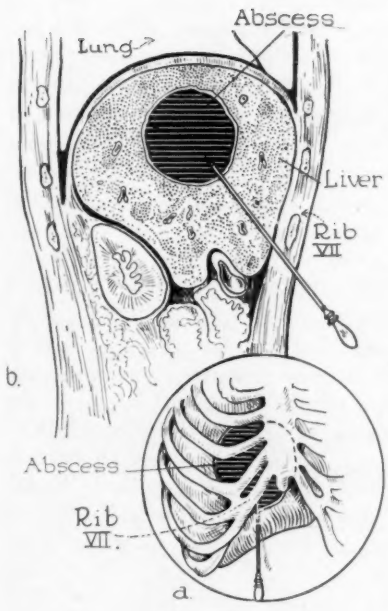


Fig. 22.

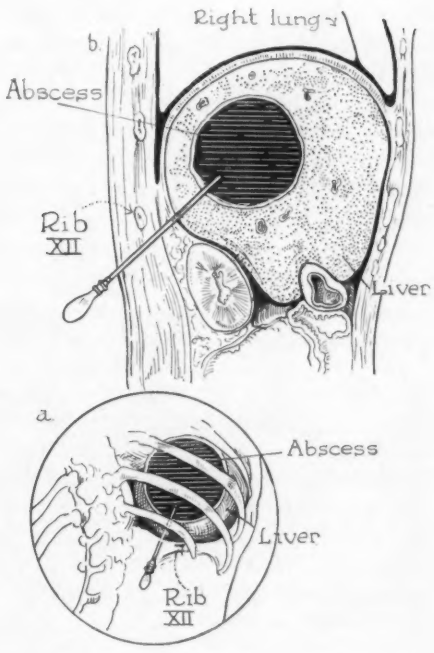


Fig. 23.

Fig. 22.—Technique of aspiration of amebic hepatic abscess located in anterior portion of liver. *a*, The needle is inserted just below the anterior costal margin about 4 to 6 cm. lateral to the midline. *b*, Diagrammatic representation of sagittal section at level of cutaneous puncture showing needle directed superiorly and posteriorly into abscess cavity.

Fig. 23.—Technique of aspiration of amebic hepatic abscess located in posterior portion of liver. *a*, The needle is inserted in the right lumbocostal angle. *b*, Diagrammatic representation of sagittal section at level of cutaneous puncture showing needle directed superiorly and anteriorly into abscess cavity.

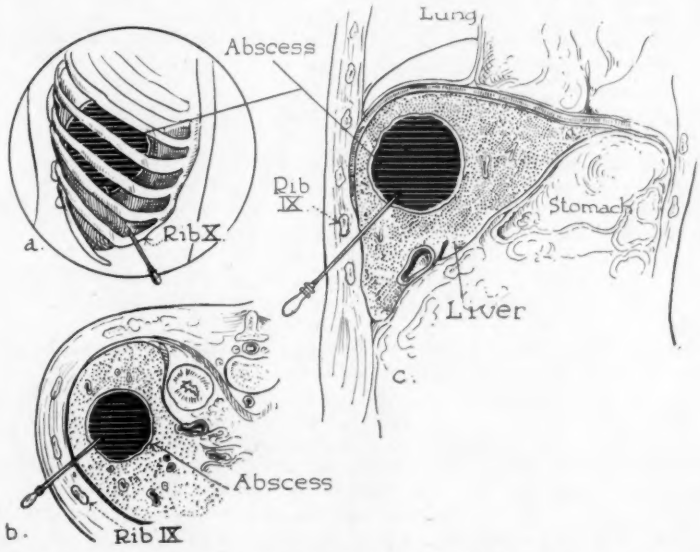


Fig. 24.—Technique of aspiration of amebic hepatic abscess located near dome of liver. *a*, The needle is inserted through the ninth or tenth intercostal space in the anterior axillary line. *b*, Composite diagrammatic representation of horizontal section through liver abscess and cutaneous puncture site showing posterior and medial direction of needle. *c*, Diagrammatic representation of sagittal section at level of cutaneous puncture showing superior direction of needle into abscess cavity.

sterile, and stool culture was negative for typhoid, paratyphoid, and bacillary dysentery. Stool examination revealed *E. histolytica* cysts. Sputum examination was negative for ameba and acid-fast organisms. Roentgenographic examination on Sept. 8, 1941, of anteroposterior and lateral views of the chest showed opacity of the right hemithorax in the lower two-thirds indicating presence of fluid in the right pleural cavity (Figs. 18 and 19). Roentgenograms taken after diagnostic thoracentesis revealed fluid level in the right pleural cavity (Fig. 20). Roentgenograms of the chest taken about one month after aspiration of chest and emetine therapy show opacity in the chest practically cleared with some elevation of the right leaf of the diaphragm (Fig. 21).

Clinical Course.—A tentative diagnosis of right bronchopneumonia with pleural effusion was made and the patient placed on sulfathiazole therapy for about five days. The temperature, which ranged around 99.6° F., gradually diminished slightly on sulfathiazole therapy but as soon as it was discontinued the temperature began to return to its former level. Following the demonstration of *E. histolytica* cysts in the stools and the aspiration of characteristic chocolate-sauce pus from the chest, the diagnosis was changed to amebic hepatic abscess with rupture into the pleural cavity and the patient was transferred to surgery. He was immediately placed on emetine therapy and another thoracentesis was performed with the removal of about 400 c.c. of typical chocolate-sauce pus. The temperature dropped to normal within 48 hours and remained there until his discharge. The clinical manifestations were also completely relieved.

REFERENCES

1. Acebal, Julio Alberto: Peritonitis generalizada por ruptura expontanea de un absceso hepatico amebiasio, An. de cir. **3**: 253, 1937.
2. Allen, W.: Medicinal Treatment of Amebic Abscess of the Liver, Surg., Gynec. & Obst. **24**: 503, 1917.
3. Alport, A. Cecil, and Ghaliougui, P.: Conservative Treatment of Liver Abscesses, Lancet **2**: 1062, 1939.
4. Anagnostopoulos, Constantin: Contribution a l'étude de la pleurésie droite symptomatique de l'abcès hépatique amebien, Presse méd. **48**: 7, 1940.
5. Andrews, Justin, and Paulson, Moses: The Incidence of Human Intestinal Protozoa With Especial Reference to Endamoeba Histolytica in the Residents of the Temperate Zone, Am. J. M. Sc. **181**: 102, 1931.
6. Annesley: Researches on the Diseases of India, London, 1828, cited by Councilman and Lafleur.[70]
7. Arrellage, Francisco C.: Absceso amebiasio de higado con trazecta Bronco-hepatico, Día Méd. **10**: 641, 1938.
8. Auvroy, M.: Quoted by Huard and Meyer-May.[174]
9. Baetjer, W. A., and Sellards, A. W.: Continuous Propagation of Amoebic Dysentery in Animals, Bull. Johns Hopkins Hosp. **25**: 165, 237, 1914.
10. Ballengal: Practical Observations of Fever Dysentery and Liver Complaint in India, Edinburgh, 1818, cited by Councilman and Lafleur.[70]
11. Barlow, W. Jarvis: Liver Abscess, South. Calif. Pract. **25**: 566, 1910.
12. Barnard, H. L.: Surgical Aspects of Subphrenic Abscess, Brit. M. J. **1**: 371, 1908.
13. Bartlett, G. B.: Pathology of Dysentery in the Mediterranean Expeditionary Force, 1915, Quart. J. Med. **10**: 185, 1917.
14. Bartlett, F. K., Corper, H. J., and Long, E. R.: The Independence of the Lobes of the Liver, Am. J. Physiol. **35**: 36, 1914.
15. Beck, C.: Subphrenic Abscess, Med. Rec. **49**: 217, 1896.
16. Beck, C.: On the Diagnosis and Treatment of Abscess of the Lung, N. York M. J. and Med. Rec. **66**: 287, 1897.
17. Béclère, A.: Sur l'aide apportée au diagnostic et la localisation des abcès dysenterique du foie par l'exploration radiologique, Bull. Soc. path. exot., Par. **1**: 79, 1908; also Arch. d'électric méd. Bordeaux **16**: 283, 1908.
18. Béclère, A.: L'exploration radiologique dans le diagnostic des affections du foie, Tribune méd. **43**: 837, 1910.
19. Bellers, A. J.: Solitary Liver Abscess: Report of 2 Cases, J. Mt. Sinai Hosp. **3**: 267, 1936-37.

20. Bernard, E.: L'amibiase pulmonaire, Médecine **10**: 366, 1929.
21. Bertrand, L. E., and Fontan, J.: Traité Médico-Chirurgical de l'hépatite suppurée des pays chauds, Soc. d'Editiones Scient. Paris, 1895.
22. Biggam, A. G., and Ghalioungui, P.: Amebic Abscess of Liver, Symptoms and Treatment With Review of 25 Cases, J. Trop. Med. **36**: 270, 1933.
23. Biggam, A. G., and Rajol, A.: Rupture of Amebic Liver Abscess Into the Peritoneal Cavity. Certain Points Concerning Its Diagnosis and Treatment, J. Trop. Med. **34**: 285, 1931.
24. Boccia, G.: Il dolore al punto sottoxifoideo (signo di Castellani) nella diagnosi differenziale tra colecistite ed epatite amebica, Riforma med. **56**: 867, 1940.
25. Boeck, W. C., and Stiles, C. W.: Studies on Various Intestinal Parasites (Especially Amoebae of Man), U. S. Hygienic Lab. Bull., No. 133, Oct., 1923, Washington Government Printing Office, 1923.
26. Boerhose: Quoted by Huard and Meyer-May.[174]
27. Boland, F. K.: Abscess of Liver, Tr. Am. S. A. **49**: 357, 1931.
28. Boland, F. K.: Surgical Complications of Amebiasis; 16 Cases, South. M. J. **98**: 7, 1936.
29. Bonnaud, P.: Quelques considerations cliniques sur les abcès du foie observes dans les pays chauds, Lyon, 1881, quoted by Huard and Meyer-May.[174]
30. Bressot: Traitement des abcès amibiens du foie par l'hépatostomie et la suture sans drainage, Progrès méd. **42**: 1150, 1927.
31. Brooks, J. D.: Abscess of the Liver, U. S. Vet. Bur. M. Bull. **4**: 363, 1928.
32. Brown, H. P. J.: Subphrenic Abscess, Ann. Surg. **14**: 260, 1927.
33. Brown, J.: Case of Great Enlargement, Suppuration, and Ulceration of the Liver in a Child Aged 12 Months, Tr. Med.-Chir. Soc. Edinburgh (1821-1825), 665, 1824.
34. Brown, Philip W., and Hodgson, Corrin H.: Late Results in Treatment of Amebic Abscess and Hepatitis of the Liver, Am. J. M. Sc. **196**: 305, 1938.
35. Buchanan, W. J.: Dysentery as a Factor in Liver Abscess. The Other Side of the Question, J. Trop. Med. **1**: 173, 1899.
36. Bucher, R.: Ueber die Ursache des bevorzugt homolateralen Sitzes des geschwulstableger und Abcessbildung in der Leber, Deutsche Ztschr. f. Chir. **143**: 209, 1934.
37. Budd, George: On Diseases of the Liver, Philadelphia, 1853, Blanchard and Lea.
38. Bunch, G. H., and Mayer, O. B.: Liver Abscess Complicated by Duodenal Fistula, South. M. J. **27**: 393, 1934.
39. Bundesen, H. N., Rawlings, I. D., and Fishbein, W. J.: The Health Hazard of Amebic Dysentery; Report of an Outbreak, J. A. M. A. **101**: 1636, 1933.
40. Candler, A. L.: Abscess of Liver Among British Eastern Troops, Lancet **1**: 429, 1920.
40a. Cantlie, James: Recovery From Liver Abscess, Followed by Abscess of the Upper Lobe of the Right Lung and Empyema, Brit. M. J. **2**: 671, 1904.
40b. Cantlie, James: Rupture of Hepatic Abscesses Into Lungs, Brit. M. J. **2**: 961, 1910.
40c. Cantlie, James: Photographs of Chests in Which Liver Pus Was Coughed Up in One Case Through the Right and in Another Through the Left Lung, J. Trop. Med. **13**: 177, 1910.
40d. Cantlie, James: Hepatic Abscess. When Pus Finds Exit by the Lung, J. Trop. Med. **13**: 248, 1910.
41. Carnot, P., and Cachéra, René: Le diagnostic et la surveillance radiologiques des grands abcès du foie par ponctions et injections aéro-lipiodolées, Arch. d. mal. de l'app. digestif. **24**: 988, 1934.
42. Carnot, P., and Cachéra, R.: Grand abcès staphylococcique du foie. Son diagnostic par ponction et injection aéro-lipiodolée, Bull. et mém. Soc. méd. d. hôp. de Paris **50**: 756, 1934.
43. Case, J. T.: The X-ray Examination of the Liver and Gall-Bladder, Arch. Roentg. Ray. **18**: 135, 1913.
44. Cassoni, T.: Sulla cura medica dell'ascesso epatico amebico, Policlinico (sez. prat.) **38**: 183, 1931.
45. Castaigne, J., and Françon, F.: L'amibiase hépatique; difficultés fréquentes de son diagnostic; valeur de l'épreuve therapeutique, J. méd. franç. **10**: 187, 1921.
46. Catteloup: Recherches sur la dysenterie du Nord de l'Afrique. Recueil de Mém. de med., etc.; Militaire, VIII, 1851.
47. Celis, A., and Medina, M.: Abscesos pulmonares amibianos apparentemente primitivos, Medicina, México **19**: 234, 1939.

48. Chang, C., and Robertson, D. S.: Amebic Abscess in Manchuria With Special Reference to Intraperitoneal Rupture, Chinese M. J. **48**: 375, 1934.
49. Chatterji, K. K.: Nonsuppurative and Suppurative Hepatitis and Splenitis of Amebic Origin, Indian J. M. **1**: 259, 1920-21.
50. Chatterji, K. K.: Surgical Aspects of Amebiasis, Indian M. Gaz. **57**: 333, 1922.
51. Chatterji, K. K.: Tropical Surgery, Baltimore, 1927, William Wood & Company.
52. Chaufford, A.: Abcès dysenterique du foie ouvert dans les bronches; guérison rapide par l'émétine, Bull. et mém. Soc. méd. d. hôp. de Paris **35**: 630, 1913.
53. Chaufford, A.: Dysentérie amibienne chronique, guérison rapide par la cure d' émétine, Bull. et mém. Soc. méd. d. hôp. de Paris **35**: 753, 1913.
54. Chaufford, A., and Clément: Grand abcès amibien du foie guéri par le traitement médical, Bull. et mém. Soc. méd. d. Hôp. de Paris **49**: 646, 1925.
55. Chen, S. M., van Gorder, S. W., and Yuan, Y. K.: Amebic Abscess of Liver, Nat. M. J. China **17**: 391, 1931.
56. Chomel: Quoted by Huard and Meyer-May.[174]
57. Chopra, R. N., Ghosh, B. N., and De, P.: Some Observations on the Toxicity of Emetine, Indian. M. Gaz. **59**: 338, 1924.
58. Chuckerbutty, S. G.: Cases Illustrative of the Pathology of Dysentery, Indian Ann. Med. Sc. **9**: 90, 1865.
59. Chang, Chi Ch'eng: Cutaneous Amebiasis Resulting From Rupture of Liver Abscess Coincident With Kala-azar, Chinese M. J. **45**: 350, 1931.
60. Chung, Huei-Lan, Khoo, F. Y., Chang, Stephen K. P., and Ma, W. S.: The Visualization of Amebic Liver Abscess, Chinese M. J. **55**: 357, 1939.
61. Cignozzi, Oresti: Traitement des abcès du foie. Observations de technique opératoire, Lyon chirr. **22**: 597, 1925.
62. Clairmont, P., and Meyer, M.: Erfahrungen über die Behandlung der Appendicitis, Acta chir. Scandinav. **60**: 55, 1926.
63. Clark, Herbert C.: The Distribution and Complications of Amebic Lesions Found in 186 Postmortem Examinations, Am. J. Trop. Med. **5**: 157, 1925.
64. Collins, M.: Two Cases of Abscess of the Liver, Southwestern Med. Gaz. **1**: 328, 1887.
65. Copher, Glover H., and Dick, Bruce, M.: "Stream line" Phenomena in the Portal Vein and the Selective Distribution of Portal Blood in the Liver, Arch. Surg. **17**: 408, 1928.
65a. Cordier: Amebiasis primitives et secondaires du poumon, Lyon méd. **133**: 730, 1924.
66. Costa: Abcès amibiens du foie, partiellement ouverts dans les bronches et dans l'intestin, guérison par les ponctions et les injections d'émétine, Bull. et mém. Soc. méd. d. hôp. de Paris **35**: 746, 1913.
67. Costantini, H.: Des indications du traitement chirurgical des abcès amibiens du foie et de la valeur de la suture primitive sans drainage, Rev. de chir. **64**: 357, 1926.
68. Costantini, H.: De l'ouverture dans les bronches des abcè amibiens du foie, Arch. méd.-chir., de l'appar. respir. **2**: 519, 1927.
69. Costantini, H., and Benhamou, E.: Abcès amibien du foie ouvert dans les voies biliaires, Mém. Acad. de chir. **65**: 601, 1939.
70. Councilman, W. T., and Lafleur, H. A.: Amebic Dysentery, Johns Hopkins Hosp. Rep. **2**: 395, 1891.
71. Couteaud: La chirurgie, l'émétine et l'epeca dans le traitement des abcès du foie, Bull. et mém. Soc. de Chir. de Paris **34**: 941, 1913.
72. Craig, Charles F.: Personal communication.
73. Craig, Charles F.: The Pathology of Chronic Specific Dysentery of Tropical Origin, Am. Med. **4**: 571, 1902.
74. Craig, Charles F.: The Etiology and Pathology of Amoebic Infection of the Intestine and Liver, Internat. Clin. **4**: 242, 1904.
75. Craig, Charles F.: The Complications of Amebic and Specific Dysentery, as Observed at Autopsy; an Analysis of 120 Cases, Am. J. M. Sc. **128**: 145, 1904.
76. Craig, C. F.: Observations Upon Amebas Infecting the Human Intestine, With a Description of Two Species, Entamoeba Coli and Entamoeba Dysenteriae, Am. Med. **9**: 854, 897, 937, 1905.
77. Craig, C. F.: The Occurrence of Endamebic Dysentery in the Troops Serving in the El Paso District From July, 1916 to December, 1916, Mil. Surgeon **40**: 286, 1917.

78. Craig, C. F.: Observation Upon the Hemolytic, Cytolytic, and Complement Binding Properties of Extracts of Amoeba Histolytica, Am. J. Trop. Med. **7**: 225, 1927.
79. Craig, C. F.: Complement Fixation in the Diagnosis of Infection With Endamoeba Histolytica, Am. J. Trop. Med. **8**: 29, 1928.
80. Craig, C. F.: The Technique and Results of a Complement Fixation Test for the Diagnosis of Infections With Endamoeba Histolytica, Am. J. Trop. Med. **9**: 277, 1929.
81. Craig, C. F.: The Diagnostic Value of the Complement Fixation Test in Amebic Infections, J. A. M. A. **95**: 10, 1930.
82. Craig, C. F.: Complement Fixation in the Diagnosis of Amebiasis, Ann. Int. Med. **5**: 170, 1931.
83. Craig, C. F.: The Amebiasis Problem, J. A. M. A. **98**: 1615, 1932.
84. Craig, C. F.: The Pathology of Amebiasis in Carriers, Am. J. Trop. Med. **12**: 285, 1932.
85. Craig, C. F.: Amebiasis and Amebic Dysentery, Springfield, Ill., 1934, Charles C. Thomas, Publisher.
86. Craig, C. F.: Observations Upon Practical Value of Complement-Fixation Test in Diagnosis of Amebiasis, Am. J. Pub. Health **27**: 689, 1937.
87. Craig, C. F., and Kagy, Edwin: A Study of Complement Fixation in Experimental Amebiasis in Dogs, Am. J. Hyg. **18**: 202, 1933.
88. Craig, C. F., and Swartzwelder, J. C.: Observations Upon Complement-Fixation Test in Monkeys Infected With Endamoeba Histolytica, Proc. Soc. Exper. Biol. & Med. **37**: 671, 1938.
89. Cunnavo, L., and Cola, S.: La diagnosi radiologica delle fistale epatobroncheoli, Radiol. med. **18**: 245, 1931.
90. Cyr, J.: Traité pratique des maladies du foie, Paris, 1887, quoted by Rolleston and McNee.[330]
91. Dale, H. H., and Dobell, Clifford: Experiments on the Therapeutics of Amoebic Dysentery, J. Pharmacol. & Exper. Therap. **10**: 399, 1917.
92. D'Antoni, J. S.: Amebiasis, Recent Concepts of Its Prevalence, Symptomatology, Diagnosis and Treatment, New Internat. Clin. **1**: 100, 1942.
93. Davidson, Andrew: Tropical Suppurative Hepatitis, in Allbutt's System of Medicine, Vol. V., New York, 1897, The Macmillan Company, pp. 134-152.
94. Davidson, Andrew: Tropical Abscess of the Liver, in Allbutt and Rolleston's System of Medicine, London and New York, 1907, Macmillan & Co., pp. 579-592.
95. DeBakey, Michael, and Ochsner, Alton: Diagnosis and Treatment of Amebic Abscess, New Orleans M. & S. J. **88**: 347, 1935.
96. deBergue: Quoted by Huard and Meyer-May.[174]
97. Déjou, L.: Traitement d'épreuve et ponction exploratrice dans le diagnostic de l'abcès tropical des foie, Monde méd. **48**: 102, 1938.
98. De Savignac: Quoted by Huard and Meyer-May.[174]
99. Dickinson, J. C.: Radiographic Findings in Hepatic Abscess, Amebic in Type, Radiology **4**: 273, 1925.
100. do Amarol, S. L.: Abcesso amebico do pulmao, Rev. clin. de São Paulo **6**: 1, 1939.
101. Dobell, Clifford: Dr. O. Uplavici (1887-1938), Parasitology **30**: 239, 1938.
102. Dobell, C., and Laidlow, P. P.: On the Cultivation of Entamoeba Histolytica and Some Other Entozoic Amoebae, Parasitology **18**: 283, 1926.
103. Dock, G.: Observations on the Amoeba Coli in Dysentery and Abscess of the Liver, Centralbl. f. Bakteriol. **10**: 227, 1891.
104. Dormer, B. A., and Friedlander, J.: Amoebiasis—Pulmonary Complications, Brit. M. J. **2**: 258, 1941.
105. Dubois, P. P.: Surveillance radiologique des abcès amibiens du foie volumineaux et antochtones à l'aide des injections d'air et de lipiodol, Paris, 1934, C. Serre, pp. 56.
106. Dutroulau: Traité des maladies des Européens dans les pays Chauds, éd. 2, Paris, 1868, J. B. Baillière.
107. Editorial: Potential Hazards of the Diagnostic Use of Thorium Dioxide, J. A. M. A. **108**: 1656, 1937.
108. Edwards, W. A., and Waterman, J. S.: Hepatic Abscess, Report of a Case With Remarks on the Amoeba Coli, Pacific M. J. **35**: 129, 1892.
109. Elkin, D. C.: Subphrenic Abscess, J. A. M. A. **97**: 1279, 1931.
110. Elliot, John B.: Abscess of the Liver, South. M. J. **8**: 1019, 1915.
111. Engman, M. F., Jr., and Meleney, H. E.: Amebiasis Cutis (Endameba Histolytica); Report of Two Cases, Arch. Dermat. & Syph. **24**: 1, 1931.

112. Eolian, R. O.: Ueber Amobenabscesse der Leber und des Subdiaphramatraums, Arch. f. klin. Chir. **163**: 137, 1930-31.
113. Eyre, E. W.: Medical Notes on Dysentery, Indian Ann. Med. Sc. **3**: 56, 1855-56.
114. Faust, E. C.: Experimental Acute Amebic Colitis in Dogs, Proc. Soc. Exper. Biol. & Med. **27**: 908, 1930.
115. Faust, E. C.: The Incidence and Significance of Infestation With Endamoeba Histolytica in New Orleans and the American Tropics, Am. J. Trop. Med. **11**: 231, 1931.
116. Faust, E. C.: Amebiasis in the New Orleans Population as Revealed by Autopsy Examination of Accident Cases, Am. J. Trop. Med. **21**: 35, 1941.
117. Faust, E. C.: Recent Laboratory Work Throwing Light on Clinical Amebiasis, Rev. Gastroenterol. **8**: 197, 1941.
118. Faust, E. C.: The Prevalence of Amebiasis in the Western Hemisphere, Am. J. Trop. Med. **22**: 93, 1942.
119. Faust, E. C., and Headler, W. H.: Intestinal Parasites. Infection of the Ambulatory White Clinic Population of New Orleans, Am. J. Trop. Med. **16**: 25, 1936.
120. Fauvel: Quoted by Huard and Meyer-May.[174]
121. Février: Quoted by Huard and Meyer-May.[174]
122. Fiessinger, N., Baudin, G., and Panayotopoulos, E.: Au sujet de l'exploration bilipiodolée des abcès du foie, Rev. méd.-chir. d. mal. du foie **11**: 337, 1936.
123. Fiessinger, N., and Casteran, R.: Le syndrome pleuro-pulmonaire de la base dans les abcès du foie, L'exploration lipiodolée des abcès du foie. Déductions de pathologie générale, Bull. et mém. Soc. méd. d. hôp. de Paris **51**: 1746, 1928.
124. Flandin, and Dumas: Gros abcès dysenterique du foie ouvert dans les bronches; guérison obtenue par le traitement chirurgical et les injections des chlorhydrate d'émétine, Bull. et mém. Soc. méd. d. hôp. de Paris **35**: 599, 1913.
125. Florence, J. J.: Quelques considérations sur l'emploi des sels d'émétine dans la dysenterie amibeinne, Province méd., Par. **24**: 470, 1913.
126. Foiret: Quoted by Huard and Meyer-May.[174]
127. Fontan: Les grands abcès du foie, Paris, 1909.
128. Françon, F.: Hépatiques amibiennes aiguës abortives, Thèse de Paris, 1919.
129. Frerichs, F. T.: A Clinical Treatise on Diseases of the Liver, Vol. II, New York, 1879, William Wood & Company, pp. 132-135.
130. Freund, H. A.: Liver Abscess, Cases Without Previous Manifestations of Amebiasis, J. A. M. A. **102**: 1550, 1934.
131. Funaro: Notes sur une nouvelle méthod exploratrice chirurgicale du foie visant specialement les abcès des pays chauds de cet organe, Égypte méd., Alexandrie, **2**: 300, 1902.
132. Futcher, T. B.: A Study of the Cases of Amebic Dysentery Occurring at the Johns Hopkins Hospital, J. A. M. A. **41**: 480, 1903.
133. Futcher, T. B., and Harvey, A. M.: Amebic Hepatitis, Internat. Clin. **3**: 1, 1937.
134. Gaide: Quoted by Huard and Meyer-May.[174]
135. Gatewood, G.: Amebic Abscess of Liver, Surgical Aspects, Illinois M. J. **71**: 41, 1937.
136. Gessner, H. B.: A Review of 96 Cases of Abscess of Liver, New Orleans M. & S. J. **85**: 793, 1933.
137. Gessner, H. B.: Liver Abscess, Am. J. Surg. **20**: 683, 1933.
138. Giffin, H. Z.: Clinical Notes on Patients From the Middle Northwest Infected With Entamebas, J. A. M. A. **61**: 675, 1913.
139. Girgis, Sami: Pulmonary Amoebiasis, J. Egyptian M. A. **22**: 402, 1939.
140. Glenard, F.: Note sur les localizations lobaires hépatiques, Bull. et mém. Soc. méd. d. hôp. de Paris **3**: 18, 386, 1901.
141. Goldburgh, H. L.: Amebic Abscess of the Liver; Report of a Case Without Previous Manifestations of Amebiasis; Operation and Recovery, Ann. Int. Med. **10**: 926, 1936.
142. Gosset, A., and Gutmann, R. A.: Nodular Hepatitis (Probable Fibrous Abscess), Un cas d'hepatite nodulaire amibienne, Bull. et mém. Soc. méd. d. hôp. de Paris **53**: 365, 1937.
143. Goulven, P.: Contribution à l'étude de l'hépatique amibienne suppurée traitée par l'émétine, sans intervention chirurgicale, Thèse de Paris, 1918-1919.
144. Granger, Amedee: Radiological Signs of Subdiaphragmatic Abscess, New Orleans M. & S. J. **82**: 748, 1930.

145. Greig, E. D. W.: Case of Amoebic Abscess of the Liver Without Antecedent Dysentery, J. Trop. Med. **43**: 119, 1940.
146. Greig, E. D. W.: Observations on a Case of Amoebic Hepatitis, J. Trop. Med. **43**: 161, 1940.
147. Greig, E. D. W.: Amoebic Abscess of the Liver Which Discharged Through the Lung, J. Trop. Med. **43**: 207, 1940.
148. González Bosch, Rodolfo: Tratamiento del absceso hepático amebiano, Día méd. **12**: 736, 1940.
149. Gravano, L., and Bianchetti, S. L.: La hepatitis supurado amebiana del lóbulo izquierdo del hígado (a propósito de dos observaciones), Semana méd. **1**: 1260, 1939.
150. Guibal, P.: Les abscès sous-phrénique, Rev. de chir. **39**: 560, 1909.
151. Haberfeld, W.: Bronchitis und peribronchitis amebiana, München. med. Wchnschr. **74**: 1834, 1927.
152. Haines, C.: Amebic Abscess, of the Liver, M. Rec. **140**: 680, 1934.
153. Hargrove, M. D.: Amebic Abscess, Treatment by Aspiration, New Orleans M. & S. J. **86**: 21, 1933.
154. Harrington, S. W.: Abscess of Left Lobe of Liver. S. Clin. North America **14**: 631, 1934.
155. Harris, H. F.: Hatfield Prize Essay, Arch. f. path. Anat. **166**: 67, 1901, quoted by Craig.[85]
156. Harris, H. F.: Pathology and Clinical History of Amebic Dysentery, J. A. M. A. **41**: 476, 1903.
157. Hartmann-Keppel, G. L.: Vingt-deux observations d'abcès amibiens du foie, traités par l'émétine, Rev. de chir. **61**: 89, 1923.
158. Harvier, P., and Maison, E.: Quoted by Huard and Meyer-May.[174]
159. Hatch, W. K.: Hepatic Abscess, Brit. M. J. **2**: 1768, 1902.
160. Hatch, W. K.: Hepatic Abscess, Lancet **2**: 1543, 1902.
161. Heathman, Lucy: Studies on the Antigenic Properties of Some Free Living and Pathogenic Amebas, Am. J. Hyg. **16**: 97, 1932.
162. Hegner, Robert, Johnson, Carl M., and Stabler, Robert M.: Host-parasite Relations in Experimental Amoebiasis in Monkeys in Panama, Am. J. Hyg. **15**: 394, 1932.
163. Herrick, A. B.: Abscess of the Liver, Surg., Gynec. & Obst. **11**: 472, 1910.
164. Hegmann, P.: De l'examen radiologique du foie, sa valeur dans les cas d'abcès, Bull. Soc. méd.-chir. de l'Indochine **4**: 364, 1913.
165. Hlava, Jaroslov: O úplavici. Predbezné sdelení Casopis lékaruv ceských., Vol. 26, No. 5, 1887; quoted by Dobell, Parasitology **30**: 239, 1938.
166. Hodgson, C. H., and Brown, P. W.: Late Results in the Treatment of Amebic Abscess of the Liver and Hepatitis, Proc. Staff Meet., Mayo Clin. **13**: 625, 1938.
167. Hodson, V. S.: The Treatment of Hepatic Abscess With Emetine, J. Trop. Med. **24**: 108, 1921.
168. Holmes, W. H.: Amebic Abscess of the Liver, Medical Treatment, Surg., Gynec. & Obst. **61**: 521, 1935.
169. Huard, P.: Nouvelles recherches sur les aspects chirurgicaux des dysenteries et colites tropicales, Arch. d. mal. de l'app. digestif. **27**: 473, 1934.
170. Huard, P.: La multiplicité et les réveils des abcès du foie, Rev. de chir. **75**: 675, 1937.
171. Huard, P., Long, M., and Graziana: Réflexions sur une nouvelle série de 48 abcès du foie, Rev. méd. franç. d'Extrême-Orient **16**: 170, 1938.
172. Huard, P., and Meyer-May, J.: Observations of 150 Cases and Autopsy Reports of 43 Cases, Bull. Soc. med.-chir. de l'Indochine **13**: 1393, 1935.
173. Huard, P., and Meyer-May, J.: Abcès du lobe hépatique gauche méconnu; péricardite puriforme aseptique consécutive; drainage trans-sternoxiphoïdien de l'abcès après repérage lipiodolé; guérison, Mém. Acad. de chir. **61**: 1343, 1935.
174. Huard, P., and Meyer-May, J.: Les Abcès du foie, Paris, 1936, Masson & Cie.
175. Huard, P., and Meyer-May, J.: One Hundred and Fifty Observations of Abscess of the Liver, Mém. Acad. de chir. **64**: 169, 1936.
176. Huard, P., and Meyer-May, J.: Traitement des abcès du foie, Bull. Soc. méd.-chir. de l'Indochine **14**: 76, 1936.
177. Huard, P., and Meyer-May, J.: 43 Autopsies d'abcès du foie, Ann. d'anat. path. **13**: 885, 1936.
178. Huard, P., and Meyer-May, J.: L'examen radiologique et la radio-chirurgie des abcès du foie, J. de chir. **47**: 529, 1936.

179. Huard, P., and Rivoolen, A.: Amibiase hépatique et cutanée à forme maligne, Rev. méd. franç. d'Extrême-Orient **17**: 1059, 1939.
180. Huard, P., and Saint-Etienne, J.: Abcès du foie probablement calcifié, Rev. méd.-chir. d. mal. du foie **14**: 278, 1939.
181. Huber: Untersuchungen über Amobendysenterie, Ztschr. f. klin. med. **67**: 262, 1909.
182. Inst. Pasteur, Hanoi, quoted by Huard and Meyer-May.[174]
183. Inst. Pasteur, Saigon, quoted by Huard and Meyer-May.[174]
184. Izar, Giudo: Epicrisi al primo caso di amebiasi cerebrale irscontracto in Italie, Minerva med. **1**: 314, 1932.
185. James, W. M.: The Effects of Certain Drugs in the Pathogenic Entamoebae of the Human Intestines, With Special Reference to the Action of Bismuth Subnitrate in Large Doses and to the Hypodermic Injection of the Hydrochloride of Emetine, Am. J. Trop. Dis. **1**: 431, 1913.
186. James, W. M.: Human Amoebiasis Due to Infection With Entamoeba Histolytica, Ann. Trop. Med. **22**: 201, 1928.
187. Jaugeas: Radioscopic Examination of the Liver, Arch. Roentg. Ray **18**: 48, 1913.
188. Jayasuriya, J. H. F.: Tropical Liver Abscess, J. Ceylon Br., Brit. M. A. **35**: 319, 1938.
189. Joslyn, H. L.: Effective Method of Treatment Amebic Abscess of the Liver, J. A. M. A. **107**: 2117, 1936.
190. Justi, K.: Colitis hyperplastica polyposa dysenterica, Arch. f. path. Anat. **234**: 31, 1921.
191. Kartulis, S.: Ueber tropische leberabse, Arch. f. path. Anat. **105**: 521, 1886.
192. Kartulis, S.: Zur Aetiology der Leberabscesse. Lebende Dysenterie-Amöben im Eiter der dysenterischen Leberabscesse, Centralbl. f. Bakteriol. u. Parasitenk. **2**: 745, 1887.
193. Kartulis, S.: Ueber tropische Leberabscesse und ihr Verhältings zur Dysenterie, Virchows Arch. f. path. Anat. **118**: 97, 1889.
194. Karunaratne, W. A. E.: The Pathology of Amoebic Abscess of the Liver, J. Ceylon Br., Brit. M. A. **35**: 167, 1938.
195. Keeton, R. W., and Hood, Marion: Pulmonary Disease Secondary to Amebiasis, M. Clin. North America **22**: 27, 1938.
196. Kessel, J. F.: Amoebiasis in Kittens Infected With Amoebae From Acute and "Carrier" Human Cases and With the Tetranucleate Amoebae of the Monkey and of the Pig, Am. J. Hyg. **8**: 311, 1928.
197. Koch, R.: Arb. a. d. Kaisere Gesundheitsamte **3**: 1, 1883.
198. Kofoid, C. A., Kornhauser, S. I., and Plate, J. T.: Intestinal Parasites in Overseas and Home Service Troops of the U. S. Army With Especial Reference to Carriers of Amebiasis, J. A. M. A. **72**: 1728, 1919.
199. Kofoid, C. A., and Swezy, Olive: On the Prevalence of Carriers of Endamoeba Dysenteriae Among Soldiers Returned From Overseas Service, Am. J. Trop. Med. **1**: 41, 1921.
200. Kovács, F.: Zur Kenntniss der Venenphänomene bei Affectionen der Tricuspidalklappe, Ztschr. f. Heilk **13**: 1, 1892.
201. Kruse, W., and Pasquale, A.: Untersuchungen über Dysenterie und Leberabscess, Ztschr. f. Hyg. u. Infectionskrankh. **16**: 1, 1894.
202. Lacaze, H., and Melnotte, P.: L'amebiase hepatique et son traitement, Rev. de chir. **66**: 709, 1928.
203. Lacroix and Vergoz: Rupture brutale d'un abcès amibien du foie dans la cavité pleurale. Opération en deux temps, Mém. Acad. de chir. **64**: 1394, 1938.
204. Lafleur, H. A.: Amoebic Dysentery. In Allbutt's System of Medicine, Vol. III, pp. 753-783; also Amoebic Abscess of the Liver, Vol. V, Pp. 153-169, New York and London, 1898, The Macmillan Company.
205. Laidlow, P., Dobell, C., and Bishop, A.: Further Experiments on the Action of Emetine in Cultures of Entamoeba Histolytica, Parasitology **20**: 207, 1928.
206. Larrey: Quoted by Huard and Meyer-May.[174]
207. Leake, C. D.: Personal communication.
208. Leake, C. D.: Chemotherapy of Amebiasis, J. A. M. A. **98**: 195, 1932.
209. LeRoy des Barres: Un cas d'abcès amibien du foie chez une enfant de huit mois, Rev. méd.-chir. d. mal. du foie **5**: 42, 1930.
210. LeRoy des Barres and Degorce: Deux radiographies d'abcès du foie, Bull. Soc. méd.-chir. de l'Indochine **1**: 49, 1910.
211. Lilienthal: Discussion of Ochsner and Graves, Ann. Surg. **98**: 961, 1933.

212. Loison: Quoted by Huard and Meyer-May.[174]
213. Loison, M.: A propos des abcès du poumon chez des dysenteriques coloniaux, Bull. et mém. Soc. de chir. **34**: 187, 1908.
214. Lösch, F.: Massenhafte Entwickelung von Amöben in Dickdarm, Arch. f. path. Anat. **65**: 196, 1875.
215. Loubejac, Armando M.: Rotura Intra-abdominal de Abcesos hepaticos Amibianos, Arch. urug. de med., cir. y especialid. **12**: 313, 1938.
216. Love, R. J. McNeill: Amebic Abscess of Liver, Brit. M. J. **1**: 696, 1918.
217. Low, George C.: A Case of Amebic Abscess of the Liver Occurring 20 Years After the Original Attack of Dysentery, Brit. M. J. **2**: 876, 1916.
218. Ludlow, A. I.: Liver Abscess. Report of 100 Operations, Surg., Gynec. & Obst. **36**: 336, 1923.
219. Ludlow, A. I.: Treatment of Abscess of the Liver by Aspiration and Subcutaneous Injection of Emetine, Chinese M. J. **38**: 93, 1924.
220. Ludlow, A. I.: Amebic Liver Abscess, Chinese M. J. **40**: 1165, 1926.
221. Luxardo, A.: Contributo clinico allo studio dell'ascesso epatico, Clin. chir. **3**: 610, 1927.
222. MacCallum, W. G.: A Textbook of Pathology, Philadelphia and London, 1932, W. B. Saunders Company.
223. MacDowall, C.: Quoted by Huard and Meyer-May.[174]
224. MacNeal, W. J., and Klenperer, P.: Amebic Abscess of the Liver, Am. J. Trop. Med. **5**: 339, 1925.
225. Macpherson: On Bengal Dysentery, Calcutta, 1850; cited by Councilman and Lafleur.[70]
226. McWhorter, G. L.: Subphrenic Abscess: An Original Extrapleural Operation, Arch. Surg. **35**: 241, 1937.
226a. Maclean, W. C.: Suppurative Inflammation of the Liver, Reynold's System of Medicine, Vol. III, p. 321, 1871.
227. Magarth, Thomas B., and Meleney, H. E.: The Complement Fixation Reaction for Amebiasis: Comparative Tests Performed by Two Laboratories, Am. J. Trop. Med. **20**: 211, 1940.
228. Magath, T. B., and Ward, C. B.: Laboratory Method of Diagnosing Amebiasis, Am. J. Hyg. **8**: 840, 1928.
229. Magendie and Pelletier: Mémoire sur l'émétine; et sur les trois espèces d'ipécacuanha, J. gen. de Méd. Chir., et pharm. **59**: 223, 1817.
230. Mallannah: Emetine and Liver Abscess, Indian M. Gaz. **47**: 331, 1913; also Brit. M. J. **1**: 1206, 1913.
231. Mallory, William J.: Solitary Amebic Abscess of the Liver, J. A. M. A. **75**: 1774, 1920.
232. Manson, Patrick: Tropical Diseases, 1°, London, 1898, Cassell & Co., Ltd., p. 353.
233. Manson-Bahr, Philip: Pulmonary Amebiasis, Lancet **2**: 599, 1923.
234. Manson-Bahr, P.: Amebic Abscess of the Liver: Its Diagnosis and Treatment. A Clinical Study, Proc. Roy. Soc. Med. (Sect. Trop. Dis. and Parasitol.) **25**: 233, 1931.
235. Manson-Bahr, P., Low, G. C., Pratt, J. J., and Gregg, A. L.: The Treatment of Liver Abscess by Aspiration With Account of 15 Cases, Lancet **1**: 941, 1923.
236. Manson-Bahr, P.: Amoebic Invasion of Skin and Subcutaneous Tissues, Tr. Roy. Soc. Trop. Med. & Hyg. **32**: 223, 1938.
237. Manson-Bahr, P.: Amoebic Dysentery and Its Effective Treatment, Brit. M. J. **2**: 255, 1941.
238. Manson-Bahr, P., and Willoughby, H.: On the Leucocyte Count in Liver Abscess, Tr. Roy. Soc. Trop. Med. & Hyg. **22**: 465, 1929.
239. Marino, S.: Clinical Study of Three Cases, Policlinico (sez. prat.) **43**: 434, 1936.
240. Matas, R.: Modern Treatment of Hepatic Abscess, Proc. Orleans Parish M. Soc., pp. 136-143, 1895.
241. Mateer, John G., Baltz, J. I., Marion, D. F., and Hollands, R. A.: The Treatment of Amebiasis by a Combined Method—Statistical End Results, Am. J. Digest. Dig. **7**: 154, 1940.
242. Megaw, J. W. D.: Some Points Connected With Liver Abscess as Seen in Calcutta, Indian M. Gaz. **40**: 81, 1905.
243. Meleney, H. E.: Community Surveys for Endamoeba Histolytica and Other Intestinal Protozoa in Tennessee, First Report, J. Parasitol. **16**: 146, 1930.
244. Meleney, H. E., Bishop, E. L., and Leathers, W. S.: Investigations of Endamoebae Histolytica and Other Intestinal Protozoa in Tennessee: III.

A State Wide Survey of the Intestinal Protozoa of Man, Am. J. Hyg. **16**: 523, 1932.

245. Melnikoff, A.: Die Chirurgischen Zugänge durch den Unteren Rand des Brustkorbes zur den Organen des Subdiaphragmaten Raumes, Deutsche Ztschr. f. Chir. **182**: 83, 1923.
246. Melnikoff, A.: Die chirurgische Anatomie der Sinus Costo-diaphragmaticus, Arch. f. klin. Chir. **123**: 133, 1923.
247. Mencher, W. H.: Amebic Abscess of the Liver, J. Mt. Sinai Hosp. **2**: 259, 1935-36.
248. Menendez, P. E.: Serological Relationship of Entamoeba Histolytica, Am. J. Hyg. **15**: 785, 1932.
249. Meyer-May, J., and Huard, P.: Quoted by Huard and Meyer-May.[174]
250. Meyer-May, J., and Huard, P.: Note sur l'étiologie des abcès du foie, Bull. Soc. path. exot. **29**: 61, 1936.
251. Merklen, Waitz, R., Albot, G., and Kabaker, J.: Hépatite amibienne à poussées ictériques répétées pendant onze ans. Abcès du foie consécutif, Bull. et mém. Soc. méd. d. hôp. de Paris **48**: 1409, 1932.
252. Miginiac, Gabriel: La vomique dans les abcès amibiens du foie. Pronostic et traitement, Rev. de chir. **60**: 100, 1922.
253. Milan, D. F., and Meleney, H. E.: Investigations of Endamoeba Histolytica and Other Intestinal Protozoa in Tennessee, Epidemaological Study of Amoebiasis in Rural Community, Am. J. Hyg. **14**: 325, 1931.
254. Miles, J. M.: The Roentgenological Diagnosis of Abscess of the Concave Surface of the Liver, Am. J. Roentgenol. **35**: 65, 1936.
255. Mondon: Notes sur des abcès du foie observés au Tonkin, Ann. d'hyg. et de méd. colon. **3**: 98, 1900.
256. Monod, C., and Vanverts, J.: De la résection du rebord costal pour la cure chirurgicale des collections sus—hépatiques, Rev. de gynéc. et de chir. abd. **1**: 499, 1897.
257. Moore: The Relation of Dysentery to Abscess of the Liver, Tr. Med. and Phys. Soc. Bombay, Vol. 8, 1862; cited by Councilman and Lefleur.[70]
258. Mouret: De la coincidence de l'hépatite ou des abscess du foie avec la dysenterie dans le pays chauds, Paris, Thèse No. 52, 1853; cited by Councilman and Lefleur.[70]
259. Moynihan, Sir B.: Abdominal Operations, Ed. 4, Vol. 2, Philadelphia, 1926, W. B. Saunders Company.
259a. Murray, F. J.: Amebic Liver Abscess, Canad. M. A. J. **26**: 312, 1932.
260. Murray, Florence J., and Koh, P. K.: Amebic Liver Abscess, Canad. M. A. J. **26**: 312, 1932.
261. Murray, R. R.: Two Unusual Cases of Abscess of the Liver, Brit. M. J. **1**: 859, 1914.
262. Musgrave, W. E.: Intestinal Amoebiasis Without Diarrhea. A Study of Fifty Fatal Cases, Philippine J. Sc. **5**: 229, 1910.
263. Musser, J. H., and Willard, Forest: A Case of Abscess of the Liver Following Amebic Dysentery, With Remarks, Univ. M. Mag., Phila. **5**: 525, 1892.
264. Nather, K., and Ochsner, A.: Retroperitoneal Operation for Subphrenic Abscess, Surg., Gynec. & Obst. **37**: 665, 1923.
265. Neligan, A. R.: The Treatment of Liver Abscess by Aspiration, Lancet **2**: 1398, 1923.
266. Nelson, E. E.: The Drugs Used in the Treatment of Amebiasis, Internat. M. Digest. **39**: 54, 1941.
267. Ngai, S. K., and Frazier, C. N.: Cutaneous Amoebiasis: A Review and a Report of Three Cases Observed in North China, Chinese M. J. **47**: 1154, 1933.
268. Niblock, W. J.: Notes on Some Interesting Surgical Cases, Indian M. Gaz. **46**: 134, 1911.
269. Ochsner, Alton: Subphrenic Abscess, New Orleans M. & S. J. **81**: 102, 1928.
270. Ochsner, Alton: Surgical Treatment of Subphrenic Infections, New Orleans M. & S. J. **82**: 752, 1930.
271. Ochsner, Alton: Subphrenic Abscess: Its Diagnosis and Treatment With Special Reference to Extraperitoneal Operation, Internat. Clin. **2**: 79, 1931.
272. Ochsner, Alton: Subphrenic Infections, Surg., Gynec. & Obst. **56**: 1095, 1933.
273. Ochsner, Alton: The Conservative Treatment of Appendiceal Peritonitis, New Orleans M. & S. J. **87**: 32, 1934.

274. Ochsner, Alton: Surgical Complications of Amebiasis, SURGERY **1**: 633, 1937.
275. Ochsner, Alton: The Conservative Treatment of Appendiceal Peritonitis, Texas State J. Med. **32**: 579, 1937.
276. Ochsner, Alton: Abdominal Wall Susceptibility to Infection, SURGERY **3**: 783, 1938.
277. Ochsner, Alton, and DeBakey, Michael: Liver Abscess. I. Amebic Abscess; Analysis of 73 Cases, Am. J. Surg. **29**: 173, 1935.
278. Ochsner, Alton, and DeBakey, Michael: Diagnosis and Treatment of Amebic Abscess of Liver (A Study Based on 4,484 Collected and Personal Cases), Am. J. Digest Dis. **2**: 47, 1935.
279. Ochsner, Alton, and DeBakey, Michael: Diseases of the Liver, Christopher, F.: A Textbook of Surgery, Philadelphia, 1936, W. B. Saunders Company, p. 1288; also pp. 1274-1286, 1942.
280. Ochsner, Alton, and DeBakey, Michael: Pleuropulmonary Complications of Amebiasis, J. Thoracic Surg. **5**: 225, 1936.
281. Ochsner, Alton, and DeBakey, Michael: Subphrenic Abscess. Collective Review. An Analysis of 3608 Collected and Personal Cases, Internat. Abstr. Surg., Surg., Gynec. & Obst. **66**: 426, 1938.
282. Ochsner, Alton, and DeBakey, Michael: Surgical Consideration of Amebiasis; Collective Review, Internat. Abstr. Surg., Surg., Gynec. & Obst. **69**: 392, 1939.
283. Ochsner, Alton, and DeBakey, Michael: Surgery of Amebiasis, New Orleans M. & S. J. **91**: 670, 1939.
284. Ochsner, Alton, and DeBakey, Michael: Surgical Amebiasis, Internat. Clin. (new series 5) **1**: 68, 1942.
285. Ochsner, Alton, DeBakey, Michael, Kleinsasser, LeRoy, and DeBakey, Ernest: Amebic Hepatitis and Hepatic Abscess, Rev. Gastroenterol. **9**: 438, 1942.
286. Ochsner, Alton, DeBakey, Michael, and Murray, Sam: Pyogenic Abscess of the Liver. Analysis of 47 Cases With Review of the Literature, Am. J. Surg. **40**: 292, 1938.
287. Ochsner, Alton, DeBakey, Michael, and Murray, Sam: Amebic Hepatic Abscess. Analysis of 139 Cases With Review of Literature, J. Internat. de Chir. **4**: 1, 1939.
288. Ochsner, Alton, and Graves, Amos: Subphrenic Abscess: An Analysis of 3,372 Collected and Personal Cases, Ann. Surg. **98**: 961, 1933.
289. Okinshevich, A. I.: Dyenterische Abscesse der Leber und ihre chirurgische Behandlung, St. Petersburg, 1911, V. S. Ettinger.
290. Opazo, G. Fernando: Diagnostico y tratamiento del absceso hepatico amibino, Rev. méd. de Chile **69**: 74, 1941.
291. Osler, W.: On the Amebae Coli in Dysentery and in Dysenteric Liver Abscess, Johns Hopkins Hosp. Bull. **1**: 53, 1890.
292. Palazuelos, Pedro Garcia, and Lerner, M. Jacabo: Diagnostico y tratamiento del absceso hepatico amebiano, Rev. méd. de Chile **69**: 67, 1941.
293. Palmer, Rex B.: Changes in the Liver in Amebic Dysentery With Special Reference to the Origin of Amebic Abscess, Arch. Path. **25**: 327, 1938.
294. Panayatalou, A., and Netter, M.: Amebiase familiale a localisations differentes. Amebiase intestinal, chez la fille. Amebiase bronchique chez la pére, Bull. et mém. Soc. méd. d. hôp. de Paris **48**: 406, 1924.
295. Pancoast, H. K.: The Roentgenological Diagnosis of Liver Abscess With or Without Subdiaphragmatic Abscess, Am. J. Roentgenol. **16**: 303, 1926.
296. Parijsky: Quoted by Melnikoff.[245]
297. Parkes, E. A.: Remarks on the Dysentery and Hepatitis of India, London, 1846; quoted by Sir Leonard Rogers, Lancet **1**: 463, 1922.
298. Pasley, Claude Burgoyne: Two Extraordinary Cases of Liver Abscess, Lancet **1**: 1076, 1915.
299. Patino Mayer, Candido, and Pataro, F. A.: Hepatitis crónica y brotes agudos con ictericia por amebiasis, Prensa méd. argent. **24**: 2139, 1937.
300. Patry: Diagnostic radiologique d'un abcès du foie, Soc. des Méd. de Syrie, Oct. 2, 1909; quoted by Huard and Meyer-May.[174]
301. Paulson, M., and Andrews, J.: Complement Fixation in Amebiasis; Comparative Evaluation in Clinical Practice, Arch. Int. Med. **61**: 562, 1938.
302. Peiris, M. V. P.: Tropical Abscess of Liver, J. Ceylon Br., Brit. M. A. **35**: 381, 1938.
303. Pellé, A.: Sur la Therapeutique de l'amibiase Pulmonaire, Paris méd. **81**: 481, 1931.

304. Perard and Roux-Berger, J.: Quarante-six cas d'abcès du foie, Bull. et mém. Soc. nat. de chir. **52**: 402, 1926.
305. Pereira Torres, Rodolfo A.: Absceso hepático amibiano. Sur patogenia, diagnóstico y tratamiento, Presna méd. argent. **27**: 2045, 2106, 1941.
306. Petridis, P.: Traitement de l'abcès tropical du foie, Bruxelles-méd. **7**: 883, 1927.
307. Petridis, P.: La forme gastrique de l'abcès du foie tropical, Lyon chir. **35**: 360, 1938.
308. Petzetakis: La broncho-amebiase. Bronchites amibiennes pures sans abcès (Presence de l'amoeba hystolytica dans les crachats), Bull. et mém. Soc. méd. d. hôp. de Par. **47**: 1229, 1923.
309. Pruvost, Pierre: Amibiase broncho-Pulmonaire. Considérations diagnostiques et thérapeutiques, Hôpital **18**: 320, 1930.
310. Purcell, F. M.: A Case of Amoebic Hepatic Abscess and Associated Pericarditis, Tr. Roy. Soc. Trop. Med. & Hyg. **31**: 689, 1938.
311. Putney, F. J., and Baker, D. C., Jr.: Amoebic Abscess of the Lung Complicated by Cerebral Abscess, Dis. of Chest **4**: 20, 1938.
312. Quadrome, C.: Ascesso del fegato apertosi nel polmone destro, importanza diagnostica della radioscopia, operazione, guarigione, Gazz. d. osp. **25**: 1497, 1901.
313. Rankmy: Quoted by Councilman and Lafleur.[70]
314. Ratcliffe, Herbert L., and Geiman, Q. M.: Spontaneous and Experimental Amebic Infection in Reptiles, Arch. Path. **25**: 160, 1938.
315. Reeves, A. J., and Apple, E. D.: Use of Thorium Dioxide in the Diagnosis of Liver Abscess, J. A. M. A. **100**: 1682, 1933.
316. Rinehart, J. J., and Anderson, H. H.: Effect of Emetine on Cardiac Muscle, Arch. Path. **11**: 546, 1931.
317. Rives and Huet: Quoted by Huard and Meyer-May.[174]
318. Rizzo, Juan: Sobre dos casos de bronco-amebiasis, Prensa méd. argent. **26**: 2139, 1939.
319. Rodolfo Varela, Ch., and Vinas Urquiza, Senero: Rotura intraperitoneal de un absceso hepatico amibiano, Rev. méd.-quir. de pat. fem. **11**: 544, 1938.
320. Rogers, Sir L.: Tropical or Amebic Abscess of the Liver and Its Relationship to Amebic Dysentery, Brit. M. J. **2**: 844, 1902.
321. Rogers, L.: The Rapid Cure of Amoebic Dysentery and Hepatitis by Hypodermic Injections of Soluble Salts of Emetine, Brit. M. J. **1**: 1424, 1912.
322. Rogers, L.: Further Experience of the Specific Curative Action in Amoebic Disease of Hypodermic Injection of Soluble Salts of Emetine, Brit. M. J. **2**: 405, 1912.
323. Rogers, L.: Sixty Cases of Amoebic Dysentery Illustrating the Treatment by Ipecacuanha and Emetine Respectively, Indian M. Gaz. **47**: 421, 1912.
324. Rogers, Sir L.: Bowel Diseases in the Tropics: Cholera, Dysenteries, Liver Abscess, and Sprue, XVI, London, 1922, Hodder & Stoughton, Ltd., pp. 475.
325. Rogers, Sir L.: Lettsonian Lectures on Amebic Liver Abscess: Its Pathology, Prevention, and Cure. Lecture I: Etiology and Pathology of Amebic Liver Abscess, Lancet **1**: 463, 1922.
326. Rogers, Sir L.: Lettsonian Lectures, etc. Lecture II. The Varieties and Treatment of Amebic Liver Abscess, Lancet **1**: 569, 1922.
327. Rogers, Sir L.: Lettsonian Lectures, etc., Lecture III. The Prevention of Amebic Liver Abscess and the Recent Reduction in Its Prevalence and Mortality, Lancet **1**: 677, 1922.
328. Rogers, Sir L.: Tropical Liver Hepatitis and Abscess, Practitioner **131**: 117, 1933.
329. Rogers, Sir L., and Megaw, J. W. D.: Tropical Medicine, Philadelphia, 1930, The Blakiston Company.
330. Rolleston, Sir H., and McNee, J. W.: Diseases of the Liver, Gallbladder and Bile Ducts, London, 1929, Macmillan & Co., Ltd.
331. Rothenberg, R. E., and Linder, W.: Single Pyogenic Liver Abscesses, 24 Cases, Surg., Gynec. & Obst. **59**: 31, 1934.
332. Rouget: Abcès amibien du foie traité par la ponction evacuatrice et les injections sans cutanées d'émétine; guérison, Bull. et mém. Soc. méd. d. hôp. de Paris **35**: 809, 1913.
333. Rouis: Recherches sur les suppurations endémiques du foie d'après des observations recueillies dans le nord de l'Afrique, pp. 456, 8°, Paris, 1860, J. B. Baillière et fils.

334. Royster, H. A., Haywood, J. B., and Stanfield, W. W.: Treatment of Amebic Abscess of Liver, Ann. Surg. **103**: 794, 1936.
335. Runyan, R. W., and Herrick, A. B.: Surgical Complications and Treatment of Intestinal Amebiasis, Am. J. Trop. Med. **5**: 137, 1925.
336. St. Johns, J. H.: The Effect of Emetine on Endamoeba Histolytica in Culture, Am. J. Hyg. **18**: 414, 1933.
337. Sambuc: Les abcès du foie à l'Hopital de Haiphong, Arch. gén. de chir. **9**: 641, 1913.
338. Sanford, A. H.: The Geographic Distribution of Amebiasis, J. A. M. A. **67**: 1923, 1916.
339. Sarmiento, Pedro J.: Absceso tropical del higado y su tratamiento con algunas innovaciones de tienica importantes, Rev. Fac. de med. **8**: 600, 1940.
340. Schrumpf-Pierron: Die chronische, periodisch aufflackernde Amöben-hepatitis, Klin. Wchnschr. **8**: 1542, 1929.
341. Sérégé, H.: Contribution à etude de la circulation du sang forte dans le foie et des localizations lobaires hepatiques, J. de méd. de Bordeaux **31**: 271, 291, 312, 1901.
342. Sérégé, H.: Etude sur le indépendence anatomique et physiologique des lobes du foie, J. de méd. de Bordeaux **32**: 327, 341, 357, 1902.
343. Sewell, E. P.: A Case of Amoebic Abscess of the Liver Which had Burst Into the Lungs, Cured by Emetine Hydrochloride, J. Roy. Army M. Corps **20**: 700, 1913.
344. Sherman, G. A., and Weinberger, H.: Amebiasis With Pleuro-pulmonary Complications; Report of 2 Cases, J. Michigan M. Soc. **40**: 289, 1941.
345. Sherwood, N. P., and Heathman, L.: Further Studies on the Antigenic Properties of Pathogenic and Free Living Amebas. II. Complement Fixation in Amebic Dysentery, Am. J. Hyg. **16**: 124, 1932.
346. Siraud: Sur un nouveau proc. pour aborder la face conveze du foie décollement et relév du cul-de-sac pleuro-diaphr. droit (vou para-pleurale transdiaphragmatique), Province méd. **15**: 603; cited by Guibal.[150]
347. Sistrunk, W. E.: Intestinal Parasites Found in Individuals Residing in the Northwest: Frequent Presence of Protozoa in Patients Who Have Never Been in Southern Countries, J. A. M. A. **57**: 1507, 1911.
348. Sitsen, A. E.: Die Komplikationen der Darmanöbiasis und deren Ursachen, Arch. f. Schiffs. u. Tropen-Hyg. **31**: 101, 1927.
349. Smith: Cited by Craig.[75]
350. Solier, L. F., and Dejou, L.: Typhlo-colites ulcéreuses inapparentes et abcès du foie amibiens sans dysenterie, Rev. de chir. **77**: 435, 1939.
351. Solonoff: First Egyptian Medical Congress, The Surgery of Abscess of the Liver, Brit. M. J. **1**: 262, 1903.
352. Spector, Bertha Kaplan: A Comparative Study of Cultural and Immunological Methods of Diagnosing Infections With Endamoeba Histolytica, J. Prev. Med. **6**: 117, 1932.
353. Spittel, R. L.: Tropical Abscess of the Liver, J. Ceylon Br., Brit. M. A. **35**: 1, 1938.
354. Staffieri, David: Las localizaciones extra-intestinales de la amibiasis, Actas y Trab. del quinto cong. nac. de med. **1**: 162, 1934.
355. Stefano, Jose: Contribución al estudio de la amebiasis pulmonar, Día méd. No. 5, 67-68, 1939 (Supp.).
356. Stengel, Alfred: Acute Dysentery and the Amoeba Coli, Med. News, Phila. **57**: 500, 1890.
357. Stevenson, W. F.: The Danger of Aspirating the Liver, Correspondence, Lancet **1**: 1428, 1898.
358. Stitt, E. R., and Curl, H. C.: Case of Liver Abscess Demonstrating the Value of a Differential Count in Diagnosis, U. States Nav. M. Bull. **4**: 376, 1910.
359. Streicher, M. H.: Amebic Abscess of Liver in Boy, Aged 7 Years, Illinois M. J. **67**: 468, 1935.
360. Strong: Cited by Rolleston and McNee.[330]
361. Strong, Richard P.: Amebic Dysentery: Abscess of Lung, Osler and Macrae System of Medicine, Vol. 1, Philadelphia, 1907, Lea Brothers and Co., p. 516.
362. Strong, R. P.: In Osler, W.: Modern Medicine, Vol. 2, Philadelphia, 1925, Lea & Febiger, p. 221.
363. Strong, R. P., and Musgrave, W. E.: Preliminary Note Regarding the Etiology of the Dysenteries of Manila, Annual Reports of the Surgeon

General of the U. S. Army, Government Printing Office, Washington, D. C., p. 251, 1900.
364. Sweet, L. K.: Amebic Abscess of the Liver, Report of a Case in a Child Five Years Old, J. Pediat. **5**: 750, 1934.
365. Tao, S. M.: Entamoebae Histolytica Infection in North China, Nat. M. J. China **17**: 412, 1931.
366. Talbot, Philip: Fifteen Cases of Liver Abscess, Brit. M. J. **2**: 375, 1919.
367. Tartavez, H.: Le décollement de la plèvre et le refoulement du diaphragm dans les interv. transthoraciques sur la face conveze du foie, Thèse de Lyon, 1897-98.
368. Tearnon, R. A.: Multiple Amebic Abscesses of Liver, Illinois M. J. **68**: 419, 1935.
369. Thierfelder, T.: Physidalischdiagnostische Vorhemerkungen zu den Leberkrankheiten; Wanderleber, Hyperämie der Leber, Perihepatitis, suppurative Hepatitis, interstitielle Hepatitis, syphilitische Hepatitis, Acute Atrophie, einfoche Atrophie und Hypertrophie der Leber, Handb. d. spec. Path. (Ziemssem) Leipz. **8**: 1, 1879, Hlft. 1 Abth., 29-280.
370. Thompson, J. E.: The Pleural and Pulmonary Complications of Tropical Abscess of the Liver, Ann. Surg. **59**: 891, 1914.
371. Thomson, J. G.: Human entamoebiasis in Temperate Zones, J. State Med. **33**: 563, 1925.
372. Thorotrast, Thorium Dioxide Preparation, J. A. M. A. **99**: 2183, 1932.
373. Thurston, E. O.: Liver Abscess, Series of 64 Cases, Lancet **2**: 1008, 1924.
374. Toullec, F., and Huard, P.: L'hémorragie grave de la ponction hépatique, Gaz. méd. de France, pp. 690, 1934.
375. Toullec, F., and Huard, P.: La ponction exploratrice du foie (dangers, indications, contraindications), Monde méd., Paris **45**: 990, 1935.
376. Touraine, A., and Duperrat, R.: L'amibiase cutanée, Presse méd. **47**: 1086, 1939.
377. Trendelenburg: Quoted by Melnikoff.[245]
378. Turner, E. L., and Beck, W. A.: Some Clinical Manifestations Resulting From Endameba Histolytica Infections, J. Tennessee M. A. **33**: 203, 1940.
379. Uplavici, O.: Paedbezne, sdelene (Ueber die Dysenterie), Centralbl. f. Bakteriol. u. Parasitenk. **1**: 537, 1887.
380. Vedder, E. B.: A Preliminary Account of Some Experiments Undertaken to Test the Efficacy of the Ipecac Treatment of Dysentery, Mil. Surgeon **29**: 318, 1911.
381. Vedder, E. B.: A Preliminary Account of Some Experiments Undertaken to Test the Efficacy of Ipecac Treatment of Dysentery, Bull. Manila Med. Soc. **3**: 48, 1911.
382. Vedder, E. B.: An Experimental Study of the Action of Ipecacuanha on Amoeba, J. Trop. Med. **15**: 313, 1912.
383. Vedder, E. B.: Origin and Present Status of the Emetine Treatment of Amebic Dysentery, J. A. M. A. **62**: 501, 1914.
384. Vergoz and Hermenjat-Gerin: De la rupture des abcès amibiens du foie dans les cavités séreuses (plèvre, péritoine, péricarde), Rev. de chir., Paris **70**: 680, 1932.
385. Vialatte, C., and Remontet, E.: Guérison par l'émétine d'un abcès du foie constitué, Bull. Soc. path. exot. **25**: 421, 1932.
386. Waring, E. J.: An Enquiry Into the Statistics and Pathology of Some Points Connected With Abscess in the Liver, as Met With in the East Indies, Trevandrum, 1854.
387. Waring, H. J.: Disease of the Liver, Gallbladder, and Biliary System, London, 1897, Macmillan & Co., Ltd., p. 91.
388. Warren, B. A.: The Diagnosis of Hepatic Abscess, U. S. Vet. Bur. M. Bull. **5**: 435, 1929.
389. Weiss, E., and Arnold, L.: Complement Fixation Test for Amebiasis, Preliminary Report, Am. J. Digest. Dis. & Nutrition **1**: 231, 1934.
390. Wenrich, D. H., Stabler, R. M., and Arnett, J. H.: Incidence of Disease-producing Amoeba (Endamoeba Histolytica) in 1060 College Freshmen and Its Significance, Science **79**: 143, 1934.
391. Wenyon, C. M., and O'Connor, F. W.: An Inquiry Into Some Problems Affecting the Spread and Incidence of Intestinal Protozoal Infections of British Troops and Natives in Egypt, With Special Reference to the Carrier Question, Diagnosis, and Treatment of Amoebic Dysentery, and an Account of Three New Human Intestinal Protozoa, J. Roy. Army Med. Corps **28**: 1, 1917.

392. Whipple, A. O.: A Study of Subdiaphragmatic Abscess, Am. J. Surg. **40**: 1, 1929.
393. Wight, Toynbee: Routine Stool Examinations for Protozoa at a United States Veterans Hospital, U. S. Vet. Bur. M. Bull. **2**: 557, 1926.
394. Wijerama, E. M.: Tropical Abscess of the Liver, J. Ceylon Br., Brit. M. A. **35**: 263, 1938.
395. Wilensky, A. O.: Amebic Liver Abscess, Case Which Perforated Into General Peritoneal Cavity; Operation and Recovery, J. Mt. Sinai Hosp. **1**: 73, 1934.
396. Wilmoth, C. L.: The Use of the Exploring Needle and Shadow Casting Media in the Diagnosis of Hepatic and Perihepatic Abscesses, Ann. Surg. **93**: 722, 1931.
397. Wilmoth, C. L.: Liver Abscess: Its Diagnosis and Treatment, J. Trop. Med. **42**: 313, 1939.
398. Woolley, P. G., and Musgrave: Bull. 32, Department of Interior, Bureau of Government Laboratories, Manilla, P. I., 1904, pp. 31.
399. Wright, H. W. S.: The Surgical Complications of Amoebic Dysentery, Brit. M. J. **2**: 261, 1941.
400. Wyatt, Thomas E., and Buchholz, Ranson R.: Amebiasis Cutis, Ann. Surg. **113**: 140, 1941.
401. Yater, W. M., and Cavanagh, J. R.: The Diagnosis of Solitary Liver Abscess by Means of Thorotrast Hepatosplenography, Ann. Int. Med. **11**: 404, 1937.
402. Yater, W. M., and Otell, L. S.: The Differential Diagnosis of Diseases of the Liver and Spleen by the Aid of Roentgenography After the Intravenous Injection of Thorotrast (Thorium Dioxide Solution), Am. J. Roentgenol. **29**: 172, 1933.
403. Young, J. R.: A Clinical Study of Liver Abscess. Report of 20 Cases, South. Surgeon **3**: 79, 1934.
404. Young, J. R., and Bristow, L. J.: Amoebic Abscess of the Liver, J. South Carolina M. A. **33**: 164, 1937.
405. Young, W. A., and Tudhope, G. R.: The Pathology of Prolonged Emetine Administration, Tr. Roy. Soc. Trop. Med. & Hyg. **20**: 93, 1926.
406. Zancarol, George: A New Operation for Hepatic Abscess, Brit. M. J. **1**: 1270, 1887.
407. Zancarol, G.: Traitement Chirurgical des abcès du foie des pays chauds, Etude clinique et experimentale, 8°, Paris, 1893.
408. Zancarol, G.: Pathogénie des abcès du foie, Rev. de chir. **8**: 8, 1893.
409. See Ref. No. 25, 138, 198, 199, 228, 338, 347, 393.
410. See Ref. No. 5, 39, 83, 114, 115, 243, 244, 253, 390.
411. See Ref. No. 6, 10, 46, 58, 63, 73, 75, 108, 113, 116, 193, 225, 257, 258, 293, 297, 313, 348, 349, 360, 363, 386, 408.
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431. See Ref. No. 47, 65a, 100, 151, 294, 308, 318, 355.

Book Reviews

Changes in the Knee Joint at Various Ages. By Granville A. Bennett, M.D., Associate Professor in Pathology, Harvard Medical School; Hans Waine, M.D., Resident Assistant in Medicine at Harvard Medical School; Walter Bauer, M.D., Associate Professor in Medicine, Harvard Medical School. Pp. 97, with 31 illustrations. New York, 1942, Commonwealth Fund Publishers.

A monograph of this sort has long been overdue. The material in this book serves as a base line from which to begin the study of pathologic material. The authors stress, particularly, the mechanistic conceptions of the origin of degenerative changes in the articular cartilage of the knee joint. There are a large number of fine plates upon which are exhibited photographs of the knee joint and microphotographs of the joint cartilage. Beginning as early as the second decade of life, and quite regularly in the fifth (after 40 years) gross evidence of degenerative changes in the articular cartilage is a regular finding. This wear and tear is a progressive phenomenon increasing in severity with added years.

This is a monograph deserving of careful study by all who profess an interest in the pathology of arthritis, be he pathologist, internist, or surgeon.

Surgical Pathology. By William Boyd, M.D., LL.D., M.R.C.P., Professor of Pathology, University of Toronto, Toronto. Ed. 5. Cloth. Pp. 843, with 520 illustrations. Philadelphia and London, 1942, W. B. Saunders Company. $10.

The author has completely revised this edition and likewise has made numerous improvements in the illustrations. It is very gratifying to the reviewer to note that he discriminates sharply between the specialties of surgical pathology and pathology; he correctly calls attention to the fact that surgical pathology involves the study of disease processes in their incipiency, whereas pathology is apt to be a study of the terminal phases of disease. However, he correctly states that a surgical pathologist should have preliminary training in pathology.

The author has adopted a clear, concise but simple style of writing. Because of this, the text lends itself very well to use by the student. The book is remarkably free from complicated discussions. The classification of tumors is simple and brief; to those trained in surgical pathology it might appear too brief but to the student, the reviewer believes, it is superior to a more inclusive classification. The author inserts sufficient material on pathogenesis and clinical manifestations to hold the reader's interest and lend proper correlation.

One of the many changes in this edition over the last one is the inclusion of carcinoma of the kidney and hypernephroma into one group, namely Hypernephroma. This change in classification might be controversial, but is an expression of the author's opinion. In general the text material represents the author's experience, but he refers constantly to the work of many other authorities to bring his material up to date.

Naturally, certain places are encountered which could be improved. The reviewer was disappointed in the author's classification of goiter. He had not adopted any consistency in the classification. Certainly the simple classification submitted years ago by the American Association for the Study of Goiter appears more descriptive

and accurate. Likewise, the classification of chronic cystic mastitis of the breast is not descriptive or consistent. The reviewer is well aware of the fact that there is no general agreement among authorities on the classification and even other features of chronic cystic mastitis.

In general the text is an admirable one, particularly for students. It reads so easily that all physicians should find it interesting. It is perhaps the best one in its field, and represents a fine addition to a medical library.

The Hand: Its Disabilities and Diseases. By C. W. Cutler, Jr., M.D., F.A.C.S., Associate Surgeon, Roosevelt Hospital, Director of Surgery, Welfare Hospital, New York City. Pp. 572, with 274 illustrations. Philadelphia and London, 1942, W. B. Saunders Company.

This book is encyclopedic in character in that it is composed chiefly of a summary of the literature, with excellent illustrations from that literature on each of the subjects considered. There appears to be, therefore, very little information original with the author.

In one sense, the book is probably more extensive in coverage than necessary. It deals not only with the infections and ailments peculiar to the hand, but also with many subjects in which consideration of the hand is only incidental, such as clubbing of the fingers in cardiorespiratory disease and the manner of taking of diverse types of skin grafts, omitting the Padgett dermatome.

On the credit side, there is good coverage of the subject of anatomy, infections, burns, wounds, fractures and dislocations, tumors, etc. There is a long list of references following each chapter, and the types of therapy outlined for the most part represent the ideas accepted by leading authorities such as Kanavel, Koch, etc.

Due to several excellent recent publications, there was no vacancy in the surgical literature requiring the preparation of this volume, but it is, nevertheless, an excellent book and one which has the advantage of presenting a wide review of the literature.

Surgery of the Ambulatory Patient. By L. K. Ferguson. Pp. 923, with 645 illustrations. Philadelphia, 1942, J. B. Lippincott Company. $10.

The author has done a good job in a comprehensive review of many surgical conditions, but the choice of the title seems a bit unfortunate. Not all the patients that come with the diseases described are ambulatory nor are all of the treated patients ambulatory, whether they are in a hospital or not. Just what patients may be ambulatory is akin to what constitutes "major" and "minor" surgery. There is danger in a book of this sort of suggesting that certain surgical procedures are of minor importance. However, as the author stresses, there is no surgical procedure that should not have careful thought given to the details before, during, and after operation.

To be sure, some doctors misuse the time and money of a patient in prolonged and unnecessary hospital stays. Especially in these times of crowded hospitals, procedures that can be safely carried out in the office and do not incapacitate the patient certainly are in order. However, unless one has the facilities and assistance, it is hazardous to attempt to do even minor surgical procedures in the average doctor's office with questionably sterile equipment and inadequate facilities in case an emergency should arise. In the reviewer's opinion, procedures that can be carried out in the office are better carried out in an operating room of a hospital. Most hospitals, in this vicinity at least, charge no more for an operative tray in the operating room than they do in the receiving ward. Whether or not

a patient is ambulatory after certain operations will depend upon the temperament of the patient and the surgeon. The trouble with most "ambulatory" surgery is that too big a job is attempted without adequate assistance and facilities.

In the main, the subjects are discussed very well. If the author had attempted to discuss fewer surgical conditions, the book would be more valuable. After all, infections on the lip are not treated generally with the patient ambulatory; also, it is questionable if one is justified in allowing a patient with an abscess of the breast, who has a fever, to be ambulatory even though the abscess is drained.

The book is profusely illustrated. The illustrations are the best the reviewer has seen in a book of this type. A comprehensive index is appended.

Abdominal and Genito-Urinary Injuries. Military Surgical Manuals, National Research Council. Pp. 243, with 28 illustrations. Philadelphia, 1942, W. B. Saunders Company.

This monograph written in two parts—one on abdominal injuries, the other injuries of the genitourinary tract—was prepared under the auspices of the Committee on Surgery of the Division of Medical Science of the National Research Council. Both portions of the volume are well done. The first section on Abdominal Injuries was written by Ambrose H. Storck; the Urological Section was written by the Subcommittee on Urology of which Herman L. Kretschmer is chairman. The latter section contains a chapter of "Do's and Don't's" for the care of urological emergencies. The illustrations in both sections are well chosen and complement the text in a very satisfactory manner.

This volume should prove a very valuable compendium for medical officers of both the Army and Navy. It can be recommended enthusiastically, too, to anyone who wishes to inform himself on the best current thought of the management of abdominal and genitourinary injuries.

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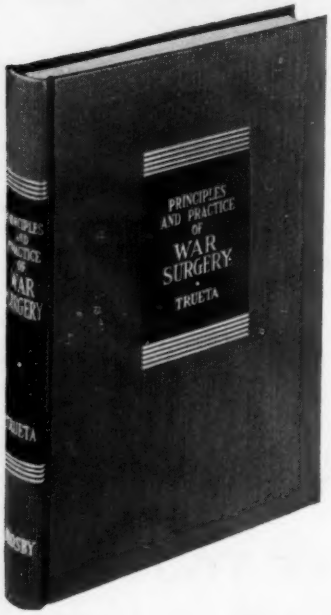

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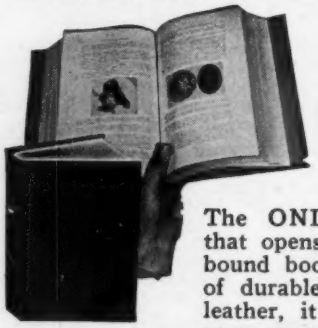

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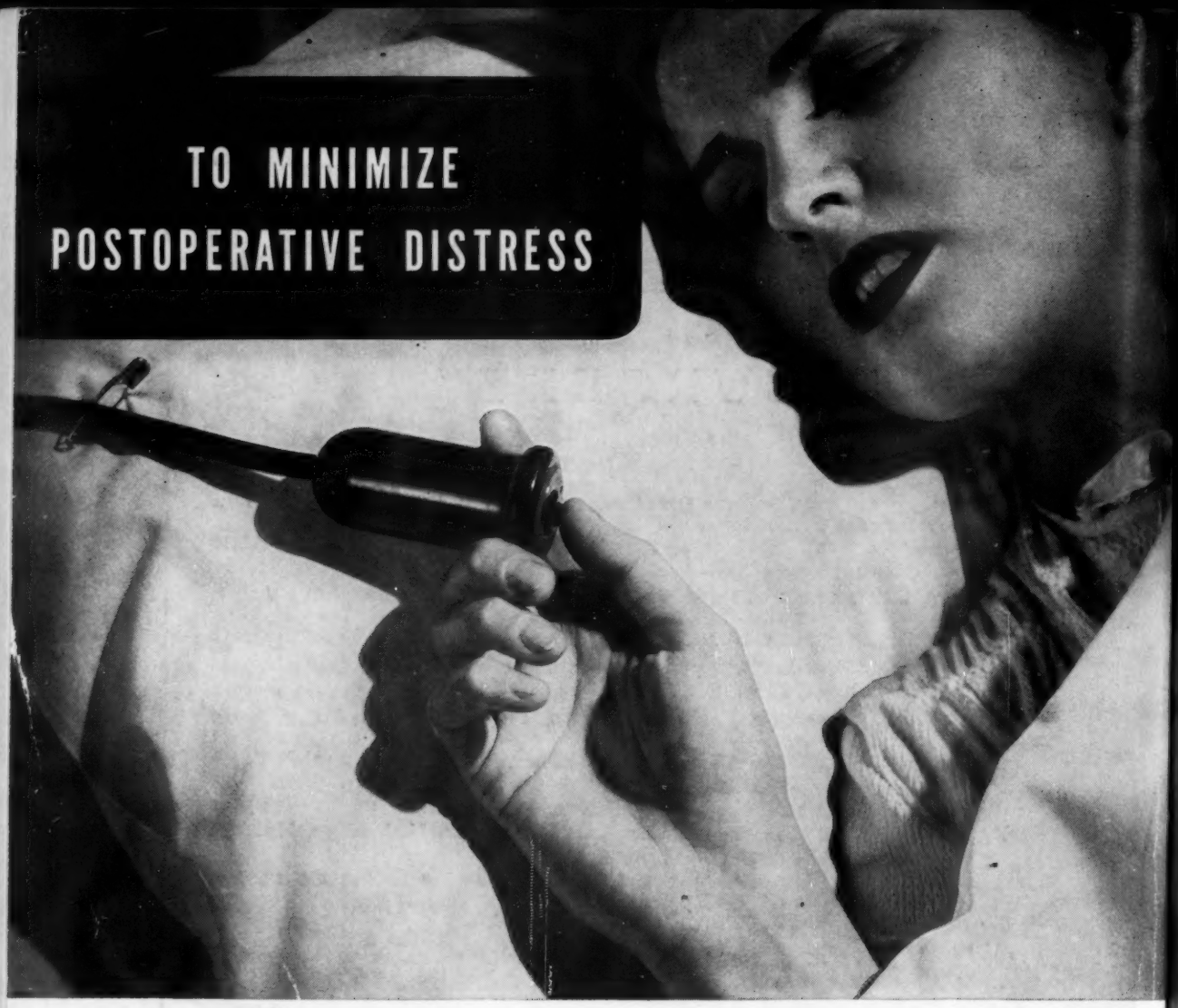

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